In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS
No. 17-989V
Filed: December 18, 2023

JASON QUIRINO,

Petitioner,

v.

SECRETARY OF HEALTH AND HUMAN SERVICES,

Respondent.

Special Master Horner

Andrew Downing, Downing, Allison & Jorgenson, Phoenix, AZ, for petitioner.
Mitchell Jones, U.S. Department of Justice, Washington, DC, for respondent.

RULING ON ENTITLEMENT[1]

On July 21, 2017, petitioner filed a petition under the National Childhood Vaccine Injury Act, 42 U.S.C. § 300aa-10, *et seq.* (2018),[2] alleging that the Hepatitis B ("Hep B") and Tetanus Diphtheria acellular Pertussis ("Tdap") vaccines he received on July 28, 2014, caused him to suffer a "rheumatologic injury." (ECF No. 1; *see also* ECF No. 35 (Amended Petition).) Petitioner later amended his petition to allege that he suffers from an "atypical form" of Guillain-Barré Syndrome ("GBS"), "manifesting as an isolated small fiber neuropathy." (ECF No. 53, p. 1.) For the reasons set forth below, I conclude that petitioner is entitled to compensation.

[1] Because this document contains a reasoned explanation for the action taken in this case, it must be made publicly accessible and will be posted on the United States Court of Federal Claims' website, and/or at https://www.govinfo.gov/app/collection/uscourts/national/cofc, in accordance with the E-Government Act of 2002. 44 U.S.C. § 3501 note (2018) (Federal Management and Promotion of Electronic Government Services). **This means the document will be available to anyone with access to the internet.** In accordance with Vaccine Rule 18(b), Petitioner has 14 days to identify and move to redact medical or other information, the disclosure of which would constitute an unwarranted invasion of privacy. If, upon review, I agree that the identified material fits within this definition, I will redact such material from public access.

[2] Within this decision, all citations to § 300aa will be the relevant sections of the Vaccine Act at 42 U.S.C. § 300aa-10-34.

I. Applicable Statutory Scheme

Under the National Vaccine Injury Compensation Program, compensation awards are made to individuals who have suffered injuries after receiving vaccines. In general, to gain an award, a petitioner must make a number of factual demonstrations, including showing that an individual received a vaccination covered by the statute; received it in the United States; suffered a serious, long-standing injury; and has received no previous award or settlement on account of the injury. Finally – and the key question in most cases under the Program – the petitioner must also establish a *causal link* between the vaccination and the injury. In some cases, the petitioner may simply demonstrate the occurrence of what has been called a "Table Injury." That is, it may be shown that the vaccine recipient suffered an injury of the type enumerated in the "Vaccine Injury Table," corresponding to the vaccination in question, within an applicable time period following the vaccination also specified in the Table. If so, the Table Injury is presumed to have been caused by the vaccination, and the petitioner is automatically entitled to compensation, unless it is affirmatively shown that the injury was caused by some factor other than the vaccination. §§ 300aa-13(a)(1)(A); 300 aa-11(c)(1)(C)(i); 300aa-14(a); 300aa-13(a)(1)(B).

In many cases, however, the vaccine recipient may have suffered an injury *not* of the type covered in the Vaccine Injury Table. In such instances, an alternative means exists to demonstrate entitlement to a Program award. That is, the petitioner may gain an award by showing that the recipient's injury was "caused-in-fact" by the vaccination in question. §§ 300aa-13(a)(1)(B); 300aa-11(c)(1)(C)(ii). In such a situation, of course, the presumptions available under the Vaccine Injury Table are inoperative. The burden is on the petitioner to introduce evidence demonstrating that the vaccination actually caused the injury in question. *Althen v. Sec'y of Health & Human Servs.*, 418 F.3d 1274, 1278 (Fed. Cir. 2005); *Hines v. Sec'y of Health & Human Servs.*, 940 F.2d 1518, 1525 (Fed. Cir. 1991).

The showing of "causation-in-fact" must satisfy the "preponderance of the evidence" standard, the same standard ordinarily used in tort litigation. § 300aa-13(a)(1)(A); *see also Althen*, 418 F.3d at 1279; *Hines*, 940 F.2d at 1525. Under that standard, the petitioner must show that it is "more probable than not" that the vaccination was the cause of the injury. *Althen*, 418 F.3d at 1279. The petitioner need not show that the vaccination was the sole cause of the injury or condition but must demonstrate that the vaccination was at least a "substantial factor" in causing the condition, and was a "but for" cause. *Shyface v. Sec'y of Health & Human Servs.*, 165 F.3d 1344, 1352 (Fed. Cir. 1999). Thus, the petitioner must supply "proof of a logical sequence of cause and effect showing that the vaccination was the reason for the injury;" the logical sequence must be supported by "reputable medical or scientific explanation, *i.e.*, evidence in the form of scientific studies or expert medical testimony." *Althen*, 418 F.3d at 1278; *Grant v. Sec'y of Health & Human Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992). A petitioner may not receive a Vaccine Program award based solely on his or her assertions; rather, the petition must be supported by either medical records or by the opinion of a competent physician. § 300aa-13(a)(1).

In what has become the predominant framing of this burden of proof, the *Althen* court described the "causation-in-fact" standard, as follows:

> Concisely stated, Althen's burden is to show by preponderant evidence that the vaccination brought about her injury by providing: (1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of proximate temporal relationship between vaccination and injury. If Althen satisfies this burden, she is entitled to recover unless the [government] shows, also by a preponderance of the evidence, that the injury was in fact caused by factors unrelated to the vaccine.

Althen, 418 F.3d at 1278 (citations omitted). The *Althen* court noted that a petitioner need not necessarily supply evidence from medical literature supporting petitioner's causation contention, so long as the petitioner supplies the medical opinion of an expert. *Id*. at 1279-80. That expert's opinion must be "sound and reliable." *Boatmon v. Sec'y of Health & Human Servs*., 941 F.3d 1351, 1359-60 (Fed. Cir. 2019) (citing *Knudsen ex rel. Knudsen v. Sec'y of Health & Human Servs.*, 35 F.3d 543, 548-49 (Fed. Cir. 1994)). The *Althen* court also indicated, however, that a Program fact-finder may rely upon "circumstantial evidence," which the court found to be consistent with the "system created by Congress, in which close calls regarding causation are resolved in favor of injured claimants." 418 F.3d at 1280.

In this case, petitioner has alleged that the Hep B and Tdap vaccines caused him to suffer an atypical form of GBS, manifesting as an isolated small fiber neuropathy. (ECF No. 53, p. 1.) Because this is not an injury listed on the Vaccine Injury Table relative to the Hep B and/or Tdap vaccines, petitioner must satisfy the above-described *Althen* test for establishing causation-in-fact.

II. Procedural History

Petitioner filed his petition on July 17, 2017, alleging that he received the Hep B and Tdap vaccinations on July 28, 2014, which caused him to develop a "rheumatologic injury." (ECF No. 1.) This case was originally assigned to Special Master Moran. (ECF No. 6.) On October 30, 2017, petitioner filed his vaccination record (Exhibit 1) and medical records (Exhibits 2-4). (ECF No. 12.) On November 9, 2017, petitioner filed his damages affidavit. (ECF No. 15 (Ex. 5).) Thereafter, the case was reassigned to Special Master Sanders. (ECF No. 19-20.)

On January 4, 2018, petitioner filed updated medical records from UCLA Health (Exhibit 6) and a statement of completion. (ECF No. 24.) On February 27, 2018, respondent filed his Rule 4(c) report, arguing that the evidence presented did not meet petitioner's burden and recommending against compensation. (ECF No. 27.) On March 26, 2018, petitioner filed additional medical records (Exhibit 7) and a response to

respondent's Rule 4(c) report. (ECF No. 29-31.) On March 27, 2018, Special Master Sanders issued a Rule 5 Order instructing petitioner to file an expert report addressing (i) petitioner's diagnosis, including whether sequela of that condition lasted for more than six months following vaccination, and (ii) a causation theory relating petitioner's diagnosis to the vaccines he received. (ECF No. 32.)

On April 9, 2018, petitioner filed an affidavit (Exhibit 8), as well as his first amended petition. (ECF Nos. 33-35.) On July 25, 2018, petitioner filed a consented motion to substitute Andrew Downing as counsel, in place of Ronald Homer. (ECF No. 38.) On November 27, 2018, petitioner filed an expert report (Exhibit 10) and curriculum vitae (Exhibit 11) of Dr. Laura S. Boylan. (ECF No. 41.) On December 17, 2018, petitioner filed his remaining medical records (Exhibit 12) and a statement of completion. (ECF No. 44-45.) On June 21, 2019, respondent filed a responsive expert report by Dr. Jeffrey Gelfand (Exhibit C), as well as Dr. Gelfand's curriculum vitae (Exhibit D) and accompanying medical literature (Exhibits E-G). (ECF No. 46.) Thereafter, this case was reassigned to my docket on August 29, 2019. (ECF No. 48-49.)

On September 5, 2019, petitioner filed medical literature (Exhibits 13-28) cited in Dr. Boylan's first expert report. (ECF Nos. 50-51.) A status conference was held on October 3, 2019, wherein I indicated that, although petitioner pled this case as a "rheumatologic injury," petitioner's expert subsequently discussed his injury of small fiber neuropathy ("SFN"). (ECF No. 52.) Related to the six-month sequela issue raised by respondent, I indicated that my preliminary view was that the medical records preponderantly evidence six months of numbness and tingling, which are consistent with Dr. Boylan's opinion of SFN. (*Id.*) Based on my review of the records, I indicated that I was preliminarily inclined to find that the onset of petitioner's alleged SFN occurred 36-hours post vaccination. (*Id.*)

Subsequently, petitioner filed his second amended petition on December 9, 2019. (ECF No. 53.) On March 3, 2020, petitioner filed a Dr. Boylan's supplemental expert report (Exhibit 29). (ECF No. 57.) On June 8, 2020, respondent filed Dr. Gelfand's supplemental expert report (Exhibit H). (ECF No. 62.) On July 14, 2020, petitioner filed a status report indicating that the matter was ready to be set for a hearing on entitlement. (ECF No. 63.)

A one-day entitlement hearing was held remotely on October 6th, 2022, via Zoom. (*See* ECF Nos. 75-86; *see also* Transcript of Proceedings ("Tr"), filed at ECF No. 90.) On November 14, 2023, petitioner filed additional authority, including a citation to an additional case that "specifically address[ed] vaccine-induced small fiber neuropathy." (ECF No. 95 (citing *Fiske v. Sec'y of Health & Human Servs.,* No. 17-1378V, 2023 WL 8352761 (Fed. Cl. Spec. Mstr. Nov. 13, 2023)).) This case is now ripe for resolution of petitioner's entitlement to compensation.

III. Factual History

a. As reflected in the medical records

On September 20, 2013, petitioner presented his primary care physician, Dr. Jamie Polito. (Ex. 2, pp. 1-4.) Physical examination was notable for normal motor and sensory function and normal reflexes. (*Id.* at 2-3.) He had a history of psoriasis and had psoriatic patches observed on the scalp and shins. (*Id.* at 1.) His psoriasis was treated topically without systemic immunosuppression. (*Id.* at 3-4.) He also had a history of rheumatic fever. (*Id.* at 1.) The primary care evaluation also recorded a complaint of low back pain, specifically "sciatica with flares." (*Id.*) Petitioner was referred to an orthopedic surgeon for further evaluation. (*Id.*)

On July 11, 2014, petitioner transferred care to a new primary care physician, Dr. Judy Kim-Hwang. (Ex. 2, p. 4.) On physical examination, there was no numbness to light touch. (*Id*. at 7.)

On July 28, 2014, he returned to Dr. Kim-Hwang for his annual physical examination. (Ex. 2, p. 9.) On review of systems, petitioner reported post nasal drip, dry cough, and episodic "sinus type" headaches, without any other neurological symptoms. (*Id*. at 9-10.) His past medical history was notable for psoriasis, irritable bowel syndrome, gastroesophageal reflux disease ("GERD"), anxiety, lattice degeneration of the retina and lumbar disc herniation. (*Id.* at 10.) On physical examination, strength was normal and there was no sensory loss to light touch. (*Id.* at 12-13.) Hemoglobin A1C was normal at 5.0, TSH was normal at 1.5, RPR was non-reactive, HIV was non-reactive, and a complete blood count (no differential), creatinine, sodium and liver function testing were normal. (*Id.* at 13-15).

At this annual appointment on July 28, 2014, petitioner received the subject Hep B and Tdap vaccinations. (Ex. 1, p. 1; Ex. 2, pp. 15.) On July 29, 2014, petitioner sent a message to Dr. Kim-Hwang, stating that he was "having a pretty rough time with [his] vaccines." (Ex. 7, p. 3.) He described how he "started feeling really sick Tuesday morning about 18 hours after the injections." (*Id.*) At this time, his symptoms included a fever, headache, and achiness in his muscles and joints, especially in his neck, shoulders, lower back, and legs. (*Id.*) On July 31, 2014, petitioner sent another message to Dr. Kim-Hwang, stating that his fever had subsided, but he had developed numbness or tingling in his hands and feet. (*Id*. at 4.) He further described how he began to experience a general feeling of exhaustion late Tuesday night. (*Id.*) Later that same day, Dr. Kim-Hwang documented a telephone note that:

> I spoke to patient this morning regarding his mychart email. He states that his fever is gone and he reports no weakness at all, though he has lethargy/no energy. Went to work and is at work presently. But since Tuesday has felt numbness in his bilateral hands (dorsal/ventral) and slightly above the wrist. No facial numbness or difficulty breathing. He has been reading about possible "autoimmune reaction/disease" that can

> develop as a result of hepatitis B vaccination and wanted to discuss that with me. I advised him that if at any point his numbness worsened or if weakness developed or any respiratory/speech issues, he should go to the ER immediately. He will see me tomorrow at 4:40PM for follow-up where I will do a complete neurological exam.

(Ex. 6, p. 2.) Later that same day, petitioner returned a call to Dr. Kim-Hwang indicating that his fever resolved, but still felt "generalized lethargy / low energy." (*Id.*) Petitioner also "agreed to see Dr. Cholfin at 9AM on 8/4/2014 for a neurology consultation regarding numbness that developed after hepatitis B and tdap vaccination." (*Id.*)

On August 1, 2014, petitioner presented to Dr. Kim-Hwang for an urgent care visit. Dr. Kim-Hwang recorded the following history:

> One day after vaccination, patient had developed fevers T 101F at home, malaise/myalgias, fatigue and onset of what patient describes as numbness/tingling in his LE/UE and now left side of the neck. . . . His fevers/myalgias subsided. Denies focal weakness or respiratory problems. Pt has been going to work and been able to work. Admittedly, he feels very depressed and anxious about this reaction as he read on the internet that the hepatitis B vaccine can cause/trigger various autoimmune conditions including MS, SLE, and [GBS].
>
> He is accompanied by his significant other who also lives with him. According to her, he has been very anxious at night losing sleep over this and thus she feels his fatigue is largely due to sleep deprivation. He admits to a long-standing hx of anxiety.
>
> He also notes that he sleeps on his stomach often with his bilateral arms crossed. He notices his symptoms most markedly at night but notices throughout the day. He describes the numbness as mild dense feeling over the tips of his fingertips and his feet, but is not consistent, comes and goes. The "tingling" sensation he describes is also a rare tingling he feels at various parts of his upper extremities. He does admit that he has known herniated disks in lumbar spine that has been recently acting up and he admits to having low back pain.
>
> He is concerned that he might be developing an autoimmune condition and this is causing him significant amount of psychological distress/anxiety. He was referred to Dr. Cholfin for evaluation and has an appointment with him on Monday. He also notes that last evening he felt a dull sensation over his left neck, which is now gone.

(Ex. 2, p. 17.) On physical examination, petitioner was afebrile, his blood pressure was 130/90, and his pulse was 74. (*Id*. at 18.) Dr. Kim-Hwang recorded normal cranial nerves; normal and symmetrical sensation to light touch, temperature, pain, and

vibratory sense; normal deep tendon reflexes; full strength; and normal gait. (*Id*.) Dr. Kim-Hwang's assessment included diagnoses of anxiety and subjective numbness/tingling. (*Id.* at 19.) Dr. Kim-Hwang informed petitioner "that it would be atypical (but not impossible) for him to develop symptoms of [GBS] so soon after vaccination." (*Id.*) It was further noted that petitioner's symptoms do not correlate with SLE and his presentation was not initially concerning for MS. (*Id.*) Petitioner was advised to see a neurologist. (*Id.*)

On August 4, 2014, petitioner presented to neurologist Dr. Jeremy Cholfin. (Ex. 2, p. 20.) Dr. Cholfin recorded the following history:

> The patient went for a routine physical appointment and received DTaP and HepC [*sic*] vaccines last Monday. The next morning he woke up with fever, aches, pains. The next day the fever broke, but he started having some unusual sensations in hands/feet, described as a hot feeling, itchy, with mild pins and needles. No frank numbness in that he could [*sic*] normal sensations, but more of a subjective sense of numbness. No weakness, balance problems, nausea, vomiting. Still has residual hand & feet numbness/tingling. Feels worse while in bed. However, he has been continuing to work, feels tired during the day, but no significant dysfunction. He is worried that it could be anxiety producing the symptoms, as he has a history of anxiety.

(*Id*.) There was no noted history of bowel or bladder symptoms. (*Id.* at 21.) On neurological examination, Dr. Cholfin recorded normal cranial nerve function; normal motor function; normal sensation to light touch, pinprick, temperature, and vibration; normal cortical sensory function; normal reflexes, including ankle reflexes; and a negative Babinski response. (*Id.* at 23 ("Downgoing toes bilaterally").) Coordination was normal, gait was normal, and the Romberg sign was negative. (*Id.*)

Dr. Cholfin's assessment was that petitioner had fever followed by "mild residual paresthesia[] and subjective numbness of the hands/feet, with a feeling of heat sensation in the feet that is bothersome at night." (Ex. 2, pp. 24-25.) Dr. Cholfin went on to write:

> This likely represents a mild vaccine reaction with subjective paresthesia[]/numbness. There is no objective evidence of deficit on exam. The patient expresses concern about whether this could represent [GBS] or MS. I provided reassurance that at this point there is no sign of either disorder. There is no history of MS symptoms, the reflexes are completely intact and there are no pathologic findings on exam that would point to either disorder.

(*Id.* at 25.) The final diagnosis was dysesthesia, and Dr. Cholfin prescribed a low dose of gabapentin at 100 mg at bedtime for symptomatic relief. (*Id.*)

On August 6, 2014, petitioner saw Dr. Kim-Hwang again in primary care. (Ex. 2, p. 26.) He indicated that his anxiety was the primary symptom that kept him up at night, causing fatigue during the day. (*Id.*) He further complained about an inability to concentrate and feelings of depression due to his belief that "he could've potentially saved himself from these symptoms by having declined the vaccinations." (*Id.*) Petitioner reported taking Xanax, "which was somewhat helpful," but he declined a prescription for Ambien "as he had difficult[ies] waking up." (*Id.*) It was noted that petitioner would "consider an antidepressant in the future;" however, in the interim, he requested a "further work-up for his symptoms." (*Id.*) Neurological examination was again recorded as normal. (*Id.* at 28.) Vitamin B12 was normal at 459. (*Id.* at 163-64.) ESR was normal at 3 and CRP was <0.3. (*Id.* at 165-66.) Dr. Kim-Hwang remarked, "Though I do not think he has a neurological disease at this present time, I do not have a definitive explanation for his symptoms at this present time." (*Id.* at 29.)

On August 12, 2014, petitioner presented to neurologist Dr. Cholfin for a follow-up. (Ex. 2, p. 30.) Petitioner reported "continued problems with tingling and numbness in the fingers and toes bilaterally" since his initial neurology consultation on August 4, 2014. (*Id.* at 31.) He further described how the numbness had spread to his lips, tongue, and neck, and how it "happened once since last visit, lasted a few hours and went away." (*Id.*) Petitioner reported feeling "tired with little energy, which is new for him." (*Id.*) He explained that, although he could still ride his bike and exercise, these activities exhausted him more than normal. (*Id.*) In addition, he reported tiredness and weakness in his thighs, arms, and shoulders, as well as "some tremor in the bilateral hands." (*Id.*) Neurological examination was again normal. (*Id.* at 32.) At this point, Dr. Cholfin concluded that "[t]he localization given the intact reflexes and both upper and lower extremity symptoms may relate to either the brain or cervical spine." (*Id.* at 34.) He ordered brain and cervical spine MRI with and without contrast but indicated that he would thereafter order electromyography and nerve conduction studies (EMG/NCS) to assess for peripheral nervous system pathology if the MRI results were not revealing. (*Id.*) Dr. Cholfin diagnosed petitioner with dysesthesia and anxiety.[3] (*Id.* at 34-35.)

An MRI of the brain on August 21, 2014, showed "[u]nremarkable contrast enhanced MRI of the brain. Small capillary telangiectasias, a benign finding. Incidental microadenoma of the pituitary gland. No intracranial abnormality visualized." (Ex. 2, p. 230.) There was no evidence of demyelinating disease. (*Id.*) An MRI of the cervical spine on that same day revealed a "[c]ircumferential disc bulge at C3-C4 with no associated spinal canal stenosis or neural foraminal stenosis. The remainder of the vertebral levels are normal. There is no abnormal enhancement in the spinal canal." (*Id.* at 229.) There were no abnormal lesions within the cervical cord. (*Id.*)

On August 25, 2014, petitioner returned to see neurologist Dr. Cholfin. (Ex. 2, p. 35.) Dr. Cholfin recorded an interval history, which noted that petitioner had been sleeping better and feeling "somewhat less anxiety" since his last visit. (*Id.*) Petitioner

[3] Dr. Cholfin remarked that "no matter what the neurologic findings are, [petitioner's] anxiety is out of proportion to the situation and [he] recommended [petitioner] seek medical attention to help manage the anxiety symptoms, as these are interfering with his life." (Ex. 2, p. 34.)

described how his shoulders and arm felt "weighted" and how the feeling was "noticeable in bed" but lessened during the day. (*Id.*) He further reported that he was still experiencing "tightness/deadening feeling in hands/fingers, feet about the same." (*Id.*) However, he stated that he was experiencing less "[b]urning dysethesias," which he had not experienced for a week. (*Id.*) Petitioner described feeling "[m]ore jittery/shaky while writing or manipulating fine objects" but having "[b]etter energy over the weekend." (*Id.*) It was noted that petitioner was running two miles every other day for exercise and playing guitar without missing notes. (*Id.*) However, petitioner was also experiencing dry mouth and tongue numbness. (*Id.*) He reported that he had "[t]ried gabapentin 100mg x1," but he "felt fatigued." (*Id.*) Neurological examination was normal with the exception of a note that sensation to light touch was "slightly reduced in finger tips, vibration slightly reduced at toes bilaterally." (*Id.* at 37.) In his assessment, Dr. Cholfin "rule[d] out peripheral neuropathy" and advised petitioner that "diagnosis of CIDP is unlikely" as "the diagnosis cannot be made in under 8 weeks after onset of symptoms." (*Id.* at 38.) However, petitioner expressed concern that he could have a mild form of CIDP. (*Id.*)

On September 4, 2014, petitioner returned to Dr. Cholfin for follow-up. (Ex. 2, p. 39.) Dr. Cholfin documented that, since his last visit, petitioner's "symptoms have been the same." (*Id.*) Petitioner again reported taking "gabapentin 100mg twice" and feeling "sleepy." (*Id.*) He stated that he had not felt like he needed it. (*Id.* at 39-40.) Petitioner reported feeling "significant residual numbness of fingers and feet" and tiredness often, although he continued to work and exercise. (*Id.* at 40.) On laboratory testing, petitioner's antinuclear antigen ("ANA") was positive at 1:80, rheumatoid factor was negative, Sjogren's antibodies were negative, and SPEP was unremarkable. (*Id.* at 42.) The neurological examination was reported as "Stable." (*Id.* at 41.) EMG/NCS on that same day revealed "[n]o electrodiagnostic evidence of a peripheral neuropathy, brachial or lumbosacral plexopathy, cervical or lumbosacral radiculopathy or irritable myopathy." (*Id.*) Dr. Cholfin's assessment was "[v]accine reaction with resulting numbness: stable to slightly improved. No evidence of medium to large fiber neuropathy on EMG/NCS." (*Id.*) He referred petitioner for rheumatological consultation. (*Id.* at 44.)

On September 15, 2014, petitioner presented to rheumatologist Dr. Geraldine Navarro. (Ex. 2, p. 44.) Dr. Navarro documented that petitioner "was in his usual state of health, until about the 28th of July - had Hep B and Tdap vax, within 24 hrs he developed chills, fevers x1 day," as well as "numbness/tingling in his LE/UE and on the L side of his neck." (*Id.* at 45.) Dr. Navarro observed that petitioner "has a lot of health anxiety" and that he was not sure whether his tingling was "due to anxiety vs a side effect of the vaccine." (*Id.*) It was further noted that petitioner had "been reading a lot on the internet" and was feeling "anxious as has read Hep B vax can lead to neuro sequelae." (*Id.*) Petitioner described a "heavy sensation in the proximal muscles of his LE and UE" that lasted "[f]or awhile." (*Id.*) He further described feeling "quite heavy" when trying to get up, feeling "a little prickly" in his feet when walking, and experiencing a constant "tightness feeling/tingling over his hand and feet." (*Id.*) Petitioner recalled that "[a]bout 4-5 days later on 8/1/14 he started to feel as though his neck, lips, mouth, and tongue if [*sic*] feels as though he is coming off the anesthetics." (*Id.*) He further

recalled taking "gabapentin x 2 pills for the hot sensation." (*Id.*) Dr. Navarro explained to petitioner that patients "with Sjogrens and SLE can present with neuropathy. His Sjogrens Abs were negative. Further recs pending ab wk up. I did explain[] that he has [a] h/o autoimmune disease, Psoriasis, and that a + ANA does not mean that he has a CTD." (*Id.* at 49.) She added that "[a]bout 5% of the normal population can have a + ANA w/o any manifestation" but ultimately agreed "that if continued symptoms he many benefit from symptomatic tx." (*Id.*)

On September 22, 2014, petitioner saw Dr. Navarro for follow-up. (Ex. 2, p. 50.) Petitioner's C3 (complement) was low; the rest of his rheumatological labs were negative other than the ANA at 1:80. (*Id.* at 53-54.) This included negative extractable nuclear antigens and antiphospholipid antibody testing. (*Id*. at 172-78.) Petitioner's CK was normal at 105. (*Id.* at 181.) Dr. Navarro recommended additional diagnostics and ordered an MRI of the spine and pelvis. (*Id.* at 53-54.) During those repeat diagnostics on October 17, 2014, petitioner's cryoglobulins were negative. (*Id.* at 188.) Repeat C3 was slightly low again at 69 (normal 76-165). (*Id.* at 187.) Petitioner also underwent an MRI of the pelvis on October 17, 2014, that revealed "[n]ormal MRI of the sacroiliac joints." (*Id.* at 231.) The MRI of the lumbar spine on that same day revealed "multilevel disc degeneration. At L4-L5 there is a small extra foraminal disc protrusion on the left which contacts the exited L4 nerve. There is also mild to moderate left foraminal narrowing at L3-L4 and L4-L5. There is no spinal canal stenosis. No abnormal enhancement is seen." (*Id.* at 232-33.)

On November 24, 2014, petitioner saw Dr. Roland Sakiyama in Internal Medicine Urgent Care and was diagnosed with pharyngitis (a sore throat) that was favored to be viral. (Ex. 2, pp. 54-58.) Petitioner was also diagnosed with headache secondary to his viral upper respiratory infection. (*Id.* at 57.)

On December 5, 2014, petitioner saw Dr. Cholfin in the neurology clinic. (Ex. 2, p. 58.) Dr. Cholfin documented that petitioner was still experiencing "persistent numbness of mid forearm to hand;" numbness in the area around his lips, tongue, and jaw; and "heat" in his hands and feet in the evening. (*Id.*) It was also noted that petitioner had started taking Lexapro two weeks prior but was no longer taking gabapentin. (*Id.*) Regarding his vaccine reaction with resulting numbness/peripheral neuropathy, Dr. Cholfin recorded that petitioner was "stable to slightly improved" and that there was "[n]o evidence of medium to large fiber neuropathy on EMG/NCS." (*Id*. at 62.) Dr. Cholfin further recorded that "ANA was mildly positive" and C3 was "mildly low." (*Id.*) It was noted that Dr. Cholfin suspected immune complex deposition[4] as a

[4] Respondent contests this assessment in his Rule 4(c) report, arguing that Dr. Cholfin "provides no support for this statement." (ECF No. 27, p. 7, n.3.) An immune complex deposition involves "the binding of antibodies to excess antigens, and subsequent deposition of the immune complexes in tissues, where they can elicit complement activation and inflammation." (*Id.*) Respondent stresses that "[t]his would require high levels of circulating antibody as well as high levels of antigenic protein," and that [t]he small quantity of protein in present-day vaccines is unlikely to cause such reactions." (*Id.*) Respondent stresses that Kurul et al. found "that a large number and frequent doses of vaccines did not lead to immune complex deposition in the choroid plexus, and did not significantly increase the deposition of immune complexes in glomeruli in unilateral nephrectomized mice." (*Id.* (citing Semra Kurul et al.,

"reaction to vaccine" but that petitioner's symptoms were "stable overall." (*Id.*) Dr. Cholfin assessed petitioner with paresthesia, dysesthesia, ANA positive, peripheral neuropathy, and "tremor, physiological." (*Id.* at 58, 62.)

On December 26, 2014, petitioner presented to Dr. Rajan Patel complaining of a cough, neck pain, and back pain. (Ex. 2, p. 63.) Petitioner did not notice any areas of weakness or numbness. (*Id.*) Moreover, petitioner told Dr. Patel that he went on a trip to Miami between December 12th through the 26th and was "moving a lot of heavy items" and subsequently noticed "some soreness on the sides of his neck going down his shoulders." (*Id.*)

On January 27, 2016, petitioner presented to Dr. Alina Katsman to establish care and with complaints of back and shoulder pain. (Ex. 2, p. 75.) Dr. Katsman noted that petitioner was suffering from right shoulder pain for about two months that "[h]appened when he was wrestling brother in Texas," and that petitioner "[s]aw Dr. Garvey on 1/16/16 for back pain." (*Id.* at 75-76.) She documented "[p]eripheral neuropathy after vaccination (Hep B and Tdap) - neuropathy mostly in hands and feet. Feet get hot; 'pins and needles' sensation." (*Id.* at 76.) She noted that petitioner saw Dr. Cholfin in 12/2014 and rheumatologist Dr. Navarro. (*Id.*) She further noted that petitioner did not take Neurontin "too much" and all of his "labs were normal except for a low C3 level and +ANA. (*Id.*)

From January 28, 2016, to June 18, 2017, petitioner treated with various medical providers, but did not report any new symptoms related to his alleged vaccine injury. (*See* Ex. 2, pp. 84-129; Ex. 4, pp. 13-30.) On June 19, 2017, petitioner was again seen by Dr. Katsman for an annual exam. (Ex. 2, p. 119.) Dr. Katsman ordered lab testing, which again showed a mildly positive ANA and a slightly low C3. (*Id.* at 221-22.) Other rheumatologic lab testing was normal. (*Id.* at 219-27.)

On August 7, 2017, petitioner presented to the UCLA Department of Orthopaedic Surgery After Hours Clinic for right knee pain after playing soccer. (Ex. 2, p. 134-38.) An MRI of the knee showed a complex medial meniscus tear and small joint effusion. (*Id.* at 236.) Thereafter, petitioner participated in physical therapy. (Ex. 4, pp. 3-10.)

Petitioner phoned UCLA Clinic on September 5, 2017, for a follow-up for atrial fibrillation, and asked for a cardiology referral. (Ex. 6, p. 24.) Petitioner's ECHO results were normal – showing no evidence of rheumatic heart disease. (*Id.* at 25.)

Petitioner presented to Dr. Malcolm Smith on December 21, 2017, for a cold that he developed while traveling in Spain. (Ex. 12, p. 22-24.) Petitioner's records reflect ongoing treatment for chronic cough that was thought to be viral, allergic and/or asthmatic. (*See id.* at 22 (interval history), 26.)

Frequent Vaccination and Immune Complex Deposition in Unilateral Nephrectomized Mice, 19 PEDIATRIC NEPHROLOGY 621 (2004) (Ex. B)).)

Petitioner's remaining records detail his office visits and emails with his treating physicians regarding his unrelated atrial fibrillation, right shoulder pain, and right knee pain. (Ex. 12, pp. 28-262.) On May 9, 2018, petitioner presented to Dr. David McAllister, complaining of right knee pain. (*Id.* at136.) Dr. McAllister noted that "[p]atient is neurologically intact in the bilateral lower extremities. Specifically, the patient is able to flex and extend the toes and ankle without any difficulty and sensation is intact to light touch along the dorsal, plantar, medial and lateral aspects of both feet." (*Id.* at 139.) Petitioner's "Problem List" continued to include peripheral neuropathy through September 7, 2018. (*See id.* at 235.) No further records were filed.

b. As reflected in petitioner's testimony

In addition to submitting an affidavit (Exhibit 8), petitioner testified at the hearing that was held in this case. (*See* Transcript of Proceedings ("Tr.") at ECF No. 90.) Petitioner testified that, prior to the date of his vaccinations, he was "an active person," who "often engaged in hiking, jogging," and bike riding. (Tr. 9-10.) He explained that, prior to the vaccinations, he had no neurological complaints. (*Id.*) Petitioner explained that he did have rheumatic fever when he was eight years old and experienced "excruciating joint pain," had a rash all over his body, and he was "mostly confined to a bed during that time." (*Id.* at 11-12.) He noted that his symptoms lasted for "the better part of a year." (*Id.* at 12.) In addition to rheumatic fever, petitioner testified that he was diagnosed with psoriasis in his late teens. (*Id.* at 13.) Petitioner described having a rash all over his body. (*Id.* at 13-14.) He testified that his psoriasis is under control and is "mostly on [his] head and face." (*Id.* at 14.) Petitioner also described his history with irritable bowel syndrome and GERD. (*Id.* at 14-16.) Petitioner explained that his father suffered from psoriasis and that his mother had rheumatic fever twice and had been diagnosed with polymyalgia rheumatica and irritable bowel syndrome. (*Id.* at 16-18.)

Petitioner testified that he received the Tdap and Hep B vaccines on July 28, 2014, when he saw Dr. Kim-Hwang. (Tr. 17-18.) He recalled that he did not have "any major concerns" during that visit. (*Id.* at 17.) Petitioner explained that he received the vaccines around 10:00 AM on a Monday, the 28th. (*Id.* at 18.) He described that at around 4:00 or 5:00 AM on July 29, 2014, he began to feel ill and developed flu-like symptoms. (*Id.*) He called his doctor the next morning and told him that he "'may have had a pretty rough time with [his] vaccinations,'" and that he "started feeling really sick Tuesday morning about 18 hours after the injections." (*Id.* at 18-19 (citing Ex. 7, p. 3).) At this point, petitioner noted that he had a fever of 101.3º and had a headache and was experiencing "really bad muscle and joint aches." (*Id.* at 19.) He described when he woke up on Wednesday, he was experiencing a numbness, stinging, tingling, tightness, and a deadening feeling in his hands and feet. (*Id.* at 20.) Petitioner testified that he called his doctor again on July 31, 2014. (*Id.* at 20-21.) During this phone call, he noted that, although his fever had resolved, he had now developed "a numbness or tingling sensation in [his] hands and feet" and he felt "exhausted, very lethargic, and no energy." (*Id.* at 21 (discussing Ex. 7, p. 4).) Petitioner clarified that he "went to bed fevery, sick on Tuesday," and developed the sensory symptoms very early Wednesday morning. (*Id.*)

Petitioner addressed the different symptoms he had throughout the course of his injury. (Tr. 22.) He stated that the sensations he had felt in his hands and his feet eventually expanded from the tips of his fingers to just below his elbows and up both of his legs to just below his knees. (*Id.*) He described that these symptoms always remained in his hands and feet but would come and go from the other areas he described. (*Id.* at 23.) Petitioner noted that he called his doctor on August 1, 2014. (*Id.*) He explained that the sensation he had been feeling in his hands and feet had now moved to his chest, neck, and head. (*Id.* at 23-24 (citing Ex. 6, p. 3).) He described how the feeling has "ebbed and flowed," but has never completely gone away. (*Id.* at 23.)

Petitioner described his first visit to his doctor following the development of his symptoms. (Tr. 25 (citing Ex. 2, p. 20).) Petitioner noted that the summary of this appointment correctly described his onset of symptoms as two days following vaccination and after his fever had already broken. (*Id.*) During this appointment, petitioner's doctor described his reaction as "a mild vaccine reaction with subjective paresthesia/numbness." (*Id.* at 26 (citing Ex. 2, p. 25).) Petitioner testified that his physician "ultimately indicated to [him] that it was caused by the vaccine, [in] his opinion," and that petitioner's condition would likely be permanent if it did not resolve within a year. (*Id.* at 26-27.) Petitioner testified that his doctor had theorized that his condition was likely due to the Hep B vaccine. (*Id.* at 27.) He noted that during that appointment, he told his doctor that he was exhausted and "had absolutely no energy." (*Id.*) During an appointment on December 5, 2014, petitioner was diagnosed with "[v]accine reaction with resulting numbness/peripheral neuropathy." (*Id.* at 53.)

Petitioner testified that his symptoms have persisted, and he still feels extremely tired. (Tr. 34-35.) He explained that his condition forced him to take days off of work here and there. (*Id.* at 35.) Petitioner testified that, in particular, there are four symptoms that have stayed with him: burning, fatigue, stinging/numbness, and itchiness. (*Id.* at 35-36.) Petitioner testified that he has not regained full ability in his hands. (*Id.* at 37.) Petitioner explained that his injury left him depressed and frightened, especially when he did not have any answers. (*Id.* at 39.)

Petitioner also addressed his medical records and the references to his injury being "related to anxiety." (Tr. 50.) He explained that he had difficulty securing an appointment and that he initially hoped "that it was just anxiety." (*Id.* at 50-51.) However, he was confident that his anxiety was not the cause of his symptoms. (*Id.* at 51-52.)

IV. Expert Opinions

a. Petitioner's expert, Laura S. Boylan, M.D.,[5] initial report, Exhibit 10

[5] Dr. Boylan has been presented without objection as an expert in neurology. (Tr. 64.) Dr. Boylan received her medical degree from Columbia University College of Physicians & Surgeons in 1994. (Ex. 11, p. 1.) She is currently an attending physician at Bellevue Hospital Medical Center in New York City,

Petitioner's expert, Dr. Boylan, opines that petitioner "more likely than not, within a reasonable degree of medical certainty," suffers from an atypical form of GBS manifesting as an isolated SFN, caused-in-fact by his Hep B and Tdap vaccinations. (Ex. 10, p. 9.) She explains that damage to small nerve fibers typically accompanies damages to larger nerve fibers, which commonly occurs in conditions like diabetic peripheral neuropathy. (*Id.* at 7.) It is also associated with GBS, hepatitis, HIV, Lyme, genetic and other systemic conditions. (*Id.* (citing J.D. England et al., *Practice Parameter: Evaluation of Distal Symmetric Polyneuropathy: Role of Autonomic Testing, Nerve Biopsy, and Skin Biopsy (An Evidence-Based Review),* 72 NEUROLOGY 177 (2009) (Ex. 15)).) She explains that GBS patients commonly experience SFN in addition to the symptoms of pain and dysesthesia typically associated with GBS. (*Id.*) Dr. Boylan cites a study by Pan et al., in which twenty patients with EMG/NCS and clinically confirmed GBS underwent skin biopsies, as well as autonomic nervous system testing. (*Id.* (citing Chun-Liang Pan et al., *Cutaneous Innervation in Guillain-Barré Syndrome: Pathology and Clinical Correlations,* 126 BRAIN 386 (2003) (Ex. 17)).) She stresses that more than half of the patients suffered small fiber nerve damage, in addition to damage in larger diameter fibers, which can be detected on routine EMG/NCS. (*Id.*)

Dr. Boylan explains that there are many variants of GBS, including twelve different forms of GBS with varying clinical and electrodiagnostic presentations in adults. (Ex. 10, p. 7 (citing Francine Vriesendorp, *Guillain-Barré Syndrome in Adults: Clinical Features and Diagnosis,* UPTODATE (Jul. 19, 2022) https://www.uptodate.com/contents/guillain-barre-syndrome-in-adults-pathogenesis-clinical-features-and-diagnosis (Ex. 19).) Some authors have suggested that isolated SFN is one such form of GBS and can similarly be caused by infection or vaccination. (*Id.* at 8 (citing U. Seneviratne & S. Gunasekera, *Acute Small Fibre Sensory Neuropathy: Another Variant of Guillain-Barré Syndrome?*, 72 J. NEUROLOGY NEUROSURGERY PSYCHIATRY 540 (2002) (Ex. 20); Nobuhiro Yuki et al., *Acute Painful Autoimmune Neuropathy: A Variant of Guillain-Barré Syndrome*, 57 MUSCLE & NERVE 320 (2018) (Ex. 21); Antonino Uncini & Nobuhiro Yuki, *Sensory Guillain-Barré Syndrome and Related Disorders: An Attempt at Systematization,* 45 MUSCLE & NERVE 464 (2012) (Ex. 22)).) Dr. Boylan highlights one study where six patients presented with acute onset numbness and dysesthesias of the arms and legs, and four were considered "post infectious." (*Id.* (citing Pan et al., *supra*, at Ex. 17).) Dr. Boylan

---

New York, and an Adjunct Professor of Neurology at the New York University School of Medicine. (Ex. 10, p. 1-2.) She also serves as an attending neurologist at the Albany Stratton VA Medical Center in Albany, New York, and works as a neurohospitalist at Essential Health in Duluth, Minnesota. (*Id.* at 2.) Dr. Boylan is board certified in neurology by the American Board of Psychiatry & Neurology. (*Id.*) She completed clinical training in internal medicine at St. Vincent's Hospital and Medical Center in New York City and completed a residency in neurology at the Neurological Institute of Columbia-Presbyterian Medical Center in New York City. (*Id.*) Dr. Boylan also completed fellowship training in Behavioral Neurology and Neuropsychiatry at the Neurological Institute of Columbia-Presbyterian Hospital in conjunction with the New York State Psychiatric Institute, both at Columbia University in New York City. (*Id.*) Dr. Boylan testified that, in her practice as a neurologist, she has treated thousands of patients with peripheral neuropathy. (Tr. 60.)

explains that "[a]nother study describes well documented cases of acute isolated small fiber neuropathy within one day to two months following vaccinations for rabies, varicella or Lyme disease." (*Id.* (citing Nizar Souayah et al., *Small Fiber Neuropathy Following Vaccination for Rabies, Varicella or Lyme Disease*, 27 VACCINE 7322 (2009) (Ex. 25)).)

Dr. Boylan stresses that patients with "one auto-immune condition are prone to develop other auto-immune conditions." (Ex. 10, p. 9.) According to Dr. Boylan, petitioner is "immunologically vulnerable." (*Id.* at 10.) Petitioner developed rheumatic fever as a young child while growing up in the suburbs of Los Angeles, California – at a time and place where it was "exceedingly rare." (*Id.* at 8-9 (citing Alberto Odio, *The Incidence of Acute Rheumatic Fever in a Suburban Area of Los Angeles,* 144 W.J. MED. 179 (1986) (Ex. 26)).) Judging by the number of hospitalizations for rheumatic fever in the area between 1971 and 1980, Dr. Boylan concludes that the overall risk of rheumatic fever at that time was "approximately 2 in a million" in suburban Los Angeles. (*Id.* (citing Odio, *supra,* at Ex. 26).) This suggests to Dr. Boylan that petitioner is especially immunologically vulnerable. (*See id.* at 10.) Moreover, petitioner's history of joint hypermobility syndrome and psoriasis "are also consistent with abnormal immune/inflammatory responses." (*Id.*)

Dr. Boylan opines that petitioner's development of atrial fibrillation following vaccination is indicative of autonomic autoimmune neuropathy. (Ex. 10, p. 9.) She explains that the autoimmune nervous system involves control of digestion, the heat, adjustment of the eyes to different lighting, emotional responses, blood pressure, and "many other 'systemic' and 'non-specific' symptoms." (*Id.*) Autonomic autoimmune neuropathy is a rare form of autoimmune neuropathy that effects only the nerves of the autonomic nervous system, "which is associated with the AchR antibody." (*Id.* (citing Martha Kerr, *Autonomic Autoimmune Neuropathy is an Antibody Mediated Disorder,* NEUROLOGY TODAY (Jan. 2004), https://journals.lww.com/neurotodayonline/Fulltext/2004/01000/Autonomic_Autoimmune_Neuropathy_Is_An.4.aspx (Ex. 27)).) Dr. Boylan explains that petitioner developed atrial fibrillation post-vaccination, which is one of a variety of cardiac problems associated with autonomic regulation. (*Id.* (citing Laura Boylan, *Brain-Heart Connections,* NEUROLOGY.ORG (Mar. 28, 2018) (Ex. 28)).)

Dr. Boylan concludes that the "time course of [petitioner's] illness is as expected in GBS." (Ex. 10, p. 10.) Petitioner had acutely acquired symptoms of non specific inflammation followed within days by a series of classical symptoms of SFN: "glove and stocking distribution of dysesthesias and pain and symptoms and signs of autonomic dysfunction including abnormal cardiac rhythms, abnormal sweating, thermoregulation and severe fatigue." (*Id.*) The syndrome started immediately following petitioner's vaccinations, which Dr. Boylan opines is "unlikely to be coincidental." (*Id.*)

b. Respondent's expert, Jeffrey M. Gelfand, M.D., M.A.S.,[6] initial report, Exhibit C

Respondent's expert, Dr. Gelfand, opines that petitioner more likely than not suffers from a "sensory syndrome," characterized by tingling, burning, itchiness, and numbness in his hands and feet, which "likely involved isolated small fiber nerves." (Ex. C, pp. 6-7.) Onset, according to Dr. Gelfand, was within one day of Hep B and Tdap vaccinations on July 24, 2014. (*Id.* at 7.) He explains that symptom onset was associated with a transient fever as well as malaise and fatigue. (*Id.*)

Upon his review of records, Dr. Gelfand observed that petitioner's neurological examinations were repeatedly normal acutely, with the exception of one abnormal exam, in which mild deficits to light touch in the fingertips and slightly reduced vibration sense in the toes was noted on Dr. Cholfin's August 25, 2019 exam, but not on his August 4th or August 12th exams. (Ex C, p. 7.) Petitioner's EMG/NCS on September 4, 2014, was normal, including no evidence of a large fiber sensory neuropathy, neuronopathy, or radiculopathy. (*Id.*) Moreover, Dr. Gelfand noted that MRI of the brain was unremarkable, including no evidence of demyelinating disease, and MRI of the cervical spine showed relatively mild degenerative changes "that would not explain [petitioner's] clinical syndrome," which Dr. Gelfand stresses is consistent with petitioner's treating neurologist's interpretation. (*Id.*) MRI of the lumbar spine showed mild degenerative changes, which Dr. Gelfand opines "would not explain his clinical syndrome (which involved his upper limbs as well)." (*Id.*)

After detailed rheumatological investigation, Dr. Gelfand observes that petitioner had a low grade positive anti-nuclear antibody and a mildly low C3 complement level on blood testing. (Ex. C, p. 7.) He opines that both results are of uncertain and "probably doubtful clinical significance – without other evidence for a systemic inflammatory / rheumatological disease except for his known history of psoriasis." (*Id.*) In Dr. Gelfand's opinion, petitioner's neurological symptoms appeared to reach a nadir "within days to a few weeks and partially improved over weeks to months, without any documented evidence of neurological relapse or progression." (*Id.*)

Dr. Gelfand opines that the record does not support a large fiber sensory neuropathy, neuronopathy, or radiculopathy as the cause of petitioner's clinical syndrome. (Ex. C, p. 7.) Instead, he explains that GBS is an acute polyradiculoneuropathy, and Brighton criteria are widely used to support this diagnosis clinically and for epidemiological purposes. (*Id.* (citing Christiaan Fokke et al.,

[6] Dr. Gelfand has been presented without objection as an expert in neurology. (Tr. 142-43.) Dr. Gelfand received his medical degree from Harvard Medical School in 2006. (Ex. D.) He completed an internship in internal medicine at University of California, San Francisco ("UCSF"), in 2007. (*Id.*) He also completed his residency in neurology, as well as a subspecialty fellowship training in multiple sclerosis and neuroimmunology, at UCSF. (*Id.*) Dr. Gelfand earned a Masters in Advanced Study (MAS) degree in Clinical Research from UCSF in 2013. (Ex. C, p. 1.) He currently serves as an attending neurologist at UCSF, and attends at both UCSF Medical Center and Zuckerberg San Francisco General Hospital, which is UCSF affiliated. (*Id.*) Dr. Gelfand specializes in caring for patients with a wide range of neuroinflammatory disorders and maintains an active clinical practice at UCSF. (*Id.*)

Diagnosis of Guillain-Barré Syndrome and Validation of Brighton Criteria, 137 BRAIN 33 (2014) (Ex. E)).) While petitioner's clinical course was monophasic and onset to nadir was more than 12 hours and perhaps not more than 28 days, "assuming that the sensory change on Dr. Cholfin's later neurological examination did not reflect true progression but rather variability of mild findings," Dr. Gelfand observes that petitioner "never had documented bilateral or flaccid weakness of his limbs; decreased or absent deep tendon reflexes in weak limbs; or nerve conduction study findings typical of GBS." (*Id.*) Dr. Gelfand stresses that these key features are needed to support a GBS diagnosis. (*Id.*) Furthermore, Dr. Gelfand suggests that EMG/NCS studies were conducted at a subacute enough timepoint following symptom onset and while petitioner was still symptomatic, which is when abnormalities would be expected if GBS was the correct diagnosis. (*Id.*) Lastly, he notes that petitioner never had a CSF examination. (*Id.*)

Dr. Gelfand acknowledges that SFN is an important diagnostic consideration, given petitioner's history of new onset tingling, burning, itching and subjective numbness in his hands and feet. (Ex. C, p. 7.) He explains that SFN refers to a neuropathy characterized by involvement of peripheral afferent unmyelinated C-fibers and thinly myelinated A-delta fibers. (*Id.* (citing Astrid Terkelsen et al., *The Diagnostic Challenge of Small Fibre Neuropathy: Clinical Presentations, Evaluations, and Causes,* 16 LANCET NEUROLOGY 934 (2017) (Ex. F)).) However, Dr. Gelfand explains that, "[u]nless large fiber nerves are involved as well as part of a mixed syndrome, isolated small fiber neuropathies will not show abnormalities" on EMG/NCS. (*Id.*) Diagnosis of isolated SFN is supported by key elements of the history and sometimes other evidence of small fiber nerve dysfunction that may be discovered by quantitative sudomotor axon reflex testing, which is "not routinely performed in clinical practice," or skin biopsy stained to evaluate for loss of intraepidermal nerve fibers. (*Id.* at 7-8 (citing Terkelsen et al., *supra,* at Ex. F).) Dr. Gelfand explains that petitioner did not have these "specialty diagnostics." (*Id.*) Based on his experience, Dr. Gelfand notes that most small fiber neuropathies present insidiously, over the course of months to years, or sometimes subacutely, over the course of weeks to months. (*Id.* at 8.) In contrast, acute-onset small fiber neuropathies are rare and not well understood. (*Id.*) According to Dr. Gelfand, autoimmune causes for acute onset SFN have been postulated in select cases, particularly in children and adolescents. (*Id.* (citing Anne Louise Oaklander, *Immunotherapy Prospects for Painful Small-Fiber Sensory Neuropathies and Ganglionopathies,* 13 NEUROTHERAPEUTICS 108 (2016) (Ex. G)).) He asserts that petitioner *did not* have other known common causes of SFN, such as diabetes, heavy alcohol use, B12 deficiency, thyroid dysfunction, chronic infection, or rheumatological disease. (*Id.*) He further explained that psoriasis is not a known or accepted cause of SFN, based on an updated search of the literature. (*Id.*)

Dr. Gelfand further acknowledges that sensory syndromes can also be a somatoform reaction in the context of anxiety. (Ex. C, p. 8.) Although he notes that this diagnosis was considered by petitioner's treating physicians, Dr. Gelfand indicates that "this would be a diagnosis of exclusion" and therefore cannot be supported, "beyond a reasonable degree of medical certainty," given the available evidence. (*Id.*)

On the question of vaccine-causation, Dr. Gelfand acknowledges that, "[a]n isolated small fiber neuropathy, which could have been immune-mediated, is a possible explanation for [petitioner's] syndrome but the evidence to support this theory does not meet the more likely than not standard [in his] opinion." (Ex. C, p. 8.) Dr. Gelfand indicates that there are no citations or peer-reviewed literature that support an association of acute-onset isolated SFN following Hep B or Tdap vaccination specifically. (*Id.* at 9.) Nor does Dr. Boylan cite any such literature. (*Id.*) Instead, Dr. Boylan cites a paper reporting an association of SFN following vaccinations for rabies, varicella, and Lyme disease. (*Id.*) Furthermore, Dr. Gelfand notes that Dr. Boylan neither provides nor cites specific evidence to support how antigens in either the Hep B vaccination or Tdap vaccination might cross-react with antigens in small fiber nerves or whether injury is thought to relate to inflammation more generally (*i.e.*, that is not antigen-directed). (*Id.*) If this was GBS or GBS-like, Dr. Gelfand stresses that clinical onset within one day would also be "too rapid [to] support a vaccine related cause of neurological injury." (*Id.*)

Even if we assume that petitioner's sensory syndrome falls within the spectrum of GBS as an atypical presentation, Dr. Gelfand stresses that the Institute of Medicine's ("IOM") report in 2012 concluded, "The epidemiological evidence is insufficient or absent to assess an association between [Tdap] and GBS." (Ex. C, p. 9 (alteration in original) (citing IOM report 2012, p. 557-58).) The IOM further concluded, "The evidence is inadequate to accept or reject a causal relationship between [Tdap] and GBS." (*Id.* (alteration in original) (citing IOM report 2012, p. 557-58).) As for the Hep B vaccine, the IOM concluded that "[t]he epidemiologic evidence is insufficient or absent to assess an association between hepatitis B vaccine and GBS . . . [t]he evidence is inadequate to accept or reject a causal relationship between hepatitis B vaccine and GBS." (*Id.* (citing IOM report 2012, p. 466).)

Therefore, Dr. Gelfand concludes that, "based on a reasonable degree of medical probability, that the Petitioner suffered from acute onset sensory syndrome with minimal objective findings on examination." (Ex. C, p. 9.) Given that (1) "[s]upporting evidence for a small fiber neuropathy, which could possibly be immune-mediated, rests in this case entirely on history without other supporting diagnostics," and (2) "[s]ymptom onset occurred within 24 hours following Tdap and Hepatitis B vaccination," there is not sufficient evidence to support that petitioner's vaccinations "more likely than not caused a possible small fiber neuropathy." (*Id.*)

c. Dr. Boylan's supplemental report, Exhibit 29

In her supplemental report, Dr. Boylan relies on a review article by Dr. Christopher Gibbons to suggest that SFN can be immune mediated. (Ex. 29, p. 1 (citing Christopher Gibbons, *Small Fiber Neuropathies,* 20 CONTINUUM 1398 (2014) (Ex. 16)).) Specifically, Dr. Boylan asserts that this article "outlines the evidence for auto-immune etiologies of small fiber neuropathy." (*Id.*)

Dr. Boylan agrees that petitioner's symptoms reached nadir within days to a few weeks of vaccination, which she explains is within the expected time frame under the Brighton criteria for a diagnosis of GBS. (Ex. 29, p. 1 (citing James J. Sejvar et al., *Guillain-Barré Syndrome and Fisher Syndrome: Case Definitions and Guidelines for Collection, Analysis, and Presentation of Immunization Safety Data,* 29 VACCINE 599 (2011) (Ex. 30)).) However, Dr. Boylan disagrees with Dr. Gelfand's opinion that petitioner's onset of symptoms was "< 1 day following vaccination." (*Id.* (citing Ex. C, p. 9).) Dr. Boylan stresses that petitioner had an "inflammatory prodrome (fever, aches, pain, malaise) within hours following the vaccine." (*Id.*) However, she emphasizes that petitioner did not report symptoms of any sensory syndrome on the same day, or within 24 hours, of vaccine administration. (*Id.* (citing Ex. 7, pp. 3-4).)

Dr. Boylan stresses that petitioner's sensory syndrome involved small fiber nerves and that a diagnosis of SFN is often made without specialty diagnostic testing. (Ex. 29, p. 1.) Dr. Boylan suggests that Dr. Gelfand either discounts or does not acknowledge (1) the conclusions of petitioner's treating neurologist Dr. Cholfin, (2) petitioner's abnormal sensory examination, (3) petitioner's abnormal complement and ANA blood tests, (4) petitioner's symptoms of dysautonomia, (5) associations of SFN with a history of autoimmune conditions, and (6) the caveats included within the Brighton criteria for GBS. (*Id.* at 1-2.) Moreover, Dr. Boylan critiques Dr. Gelfand's report because he "does not offer an explicit opinion as to an alternate diagnosis." (*Id.* at 2.) Rather, Dr. Gelfand suggests that a somatoform, or psychological, disorder is more likely than SFN. (*Id.*) Dr. Boylan stresses that this opinion lacks analysis or explanation, beyond the fact that such a diagnosis was entertained by some of petitioner's treating physicians. (*Id.*) In fact, Dr. Boylan stresses that SFN has been increasingly associated with conditions "long considered by many in the medical community as being of psychological in origin," such as fibromyalgia, chronic fatigue syndrome, and complex regional pain syndrome. (*Id.* (citing Gibbons, *supra*, at Ex. 16; Terkelsen et al., *supra*, at Ex. F; Anne Louise Oaklander & Maria Nolano, *Scientific Advances in and Clinical Approaches to Small-Fiber Polyneuropathy: A Review*, 76 JAMA NEUROLOGY 1135 (2019) (Ex. 32)).)

Dr. Boylan cites one study involving a poly-ethnic U.S. sample of juveniles with unexplained widespread pain. (Ex. 29, p. 2 (citing Anne Louise Oaklander & Max M. Klein, *Evidence of Small-Fiber Polyneuropathy in Unexplained, Juvenile-Onset, Widespread Pain Syndromes,* 131 PEDIATRICS e1091 (2013) (Ex. 33)).) Physical examinations revealed no neurological abnormalities and electrodiagnostic studies were noncontributory, except in the one case of a patient with a prior brachial plexitis. (*Id.* (citing Oaklander & Klein, *supra*, at Ex. 33, p. 5).) Dr. Boylan notes that SFN was identified as definite by clinical history and objective testing in 59% of this study group and an additional 39% were considered to have possible or probable SFN. (*Id.* (citing Oaklander & Klein, *supra*, at Ex. 33, p. 5).) Furthermore, a history of auto-immune disease was identified in 33% of 41 patients. (*Id.* (citing Oaklander & Klein, *supra*, at Ex. 33, pp. 1, 5).) Dr. Boylan emphasizes that petitioner has a history of auto-immune diseases (*i.e.*, rheumatic fever and psoriasis). (*Id.*) In the Oaklander & Klein study, a wide range of autoimmune conditions were associated with small fiber polyneuropathy.

(*Id.* (citing Oaklander & Klein, *supra*, at Ex. 33, p. 5).) Dr. Boylan stresses that, like petitioner, three participants had joint hypermobility (*i.e.*, Ehlers-Danlos Syndrome). (*Id.* (citing Oaklander & Klein, *supra*, at Ex. 33, p. 5).)

Dr. Boylan suggests that the Brighton criteria were developed for use in research studies and exclude most clinical variants of GBS. (Ex. 29, p. 2 (citing Gibbons, *supra*, at Ex. 16; Vriesendorp, *supra*, at Ex. 19; James Sejvar et al., *supra*, at Ex. 30).) She explains that the Brighton criteria caution that case definitions differ based on "currently available data, intended use and clinical setting." (*Id.*) Moreover, the Brighton criteria were designed to be strict, or "specific," and Dr. Boylan contends that the criteria may miss up to 1% of GBS cases. (*Id.*) She adds that "[i]t is recognized by the group that the case definitions for GBS and Fisher Syndrome may not capture some of the clinical variants that nonetheless may be related and are regarded by others to be forms of 'GBS.'" (*Id.* (quoting Vriesendorp, *supra*, at Ex. 19).)

Dr. Boylan disagrees with Dr. Gelfand's suggestion that a somatoform reaction is a possible explanation of petitioner's condition. (Ex. 29, pp. 2-3.) She claims that Dr. Gelfand's statement that a somatoform diagnosis is a diagnosis of exclusion is not in line with the current thinking of leading neurologists with a special interest in this area. (*Id.* at 3 (citing Jon Stone, *Conversion Disorders in Adults: Clinical Features, Assessment, and Comorbidity,* UPTODATE (Jan. 7, 2020) (Ex. 35); Jon Stone, *Conversion Disorder in Adults: Terminology, Diagnosis, and Differential Diagnosis,* UPTODATE (Jan. 7, 2020) (Ex. 36); Alberto J. Espay et al., *Current Concepts in Diagnosis and Treatment of Functional Neurological Disorders,* 75 JAMA NEUROLOGY 1132 (2018) (Ex. 37)).) Instead, Dr. Boylan explains that, when somatoform reactions take the form of specific neurological conditions, they may be referred to as "functional neurological disorders." (*Id.* (citing Stone, *supra*, at Ex. 36).) According to Dr. Boylan, "the area of functional neurological disorders involves inclusionary as well as exclusionary features and does not rely on the presence or absence of psychological stressors or suggestive historical clues." (*Id.* (citing Stone, *supra*, at Ex. 36; Espay et al., *supra*, at Ex. 37).) In the case of sensory syndromes, Dr. Boylan opines that inclusionary criteria include specific findings on the physical examination, such as "precise splitting of the midline on vibrations sensation on the forehead or sternum, sharply demarcated sensory loss at the groin or shoulder and certain abnormalities on the testing of visual fields." (*Id.*) She adds that there are additional "findings on physical examination seen with functional neurological disorders which extend beyond sensory syndromes to include problems with movement and strength." (*Id.*) She stresses that the diagnosis of functional neurological disorders in petitioner's case is "based on broad principles of inconsistency of symptoms over time and phenomenology and incongruence of symptoms with known neurological disorders." (*Id.*) Dr. Boylan suggests that "[a]nxiety is ubiquitous in medical practice, particularly among patients with unquestionably biologically based neurological or other medical illnesses." (*Id.*)

In her clinical experience, Dr. Boylan contends that the "[f]ixed classical symptoms" of SFN, in the absence of electrodiagnostic findings and/or established peripheral neuropathy that is stable over years of time, is not common in clinical

practice. (Ex. 29, p. 3.) In her opinion, petitioner acquired an atypical form of GBS, manifesting as an isolated SFN, that was caused-in-fact by his Hep B and Tdap vaccines. (*Id.* at 4.)

d. Dr. Gelfand's supplemental report, Exhibit H

In his supplemental report, Dr. Gelfand recalls that petitioner developed fever, aches, and pain within 18 hours after vaccination. (Ex. H, pp. 2-3.) This was followed by sensory complaints that appeared as early as Tuesday evening (July 29, 2014), which Dr. Gelfand explains would put the onset of sensory symptoms between 24-48 hours postvaccination. (*Id.* (citing Ex. 7, p. 4; Ex. 6, p. 2; Ex. 2, pp. 17, 20).) Dr. Gelfand suggests that if you include "the symptom complex as a whole (fevers, aches, pain followed shortly after by sensory symptoms)," then symptom onset began as early as about 18 hours post-vaccination. (*Id.* at 3.) However, if you exclude fevers/aches/pains and only include sensory symptoms (as Dr. Boylan proposes), then onset of was about 24-48 hours post-vaccination. (*Id.*) Dr. Gelfand concludes that a timeframe of between 18-48 hours following vaccination "would be very quick to cause a Guillain-Barre type syndrome." (*Id.*)

Dr. Gelfand acknowledges that small fiber neuropathies may be considered "Guillain-Barre-like" in that they resemble GBS "in time course and by being immune-mediated;" however, he stresses that small fiber neuropathies "are best understood as stand-alone conditions in terms of epidemiology and immunology." (Ex. H, p. 3.) He also acknowledges that Oaklander and Nolano and "others have proposed the existence of small fiber–targeting inflammatory SFN, with acute and chronic presentations temporally resembling Guillain-Barre syndrome and chronic inflammatory demyelinating polyneuropathy, and preliminary evidence of episodic relapsing-remitting courses." (*Id.* (quoting Oaklander & Nolano, *supra*, at Ex. 32, p. 7).) However, Dr. Gelfand explains that, unlike in GBS and chronic inflammatory demyelinating polyneuropathy, "inflammatory cells are not prominent in SFN biopsies or cerebrospinal fluid." (*Id.* (quoting Oaklander & Nolano, *supra*, at Ex. 32, p. 7).) Dr. Gelfand also contends that none of the 12 GBS variants listed in Vriesendorp's article include "pure small fiber neuropathy," and petitioner never had absent reflexes or other evidence of a concurrent large fiber neuropathy. (*Id.* (citing Vriessendorp, *supra*, at Ex. 19).) Furthermore, he stresses that none of the medical literature supports an association of an isolated SFN, specifically observed or reported with Hep B or Tdap vaccination, nor is there specific evidence to support how antigens in either of those vaccinations might cross-react with antigens in small fiber nerves. (*Id.*)

e. Dr. Boylan's in court testimony

Dr. Boylan testified at the hearing on behalf of petitioner. (Tr. 54.) She defined neuropathy as "a general term for injury to nerves," which can include large, medium, and small fibers. (*Id.* at 64.) Small fiber nerves specifically "deal with itching sensation, pain, burning, [or] the types of things we generally refer to as neuropathic pain." (*Id.* at 67.) Dr. Boylan explained that peripheral neuropathy is diagnosed through a review of

the patient's history and physical examination, as well as through an EMG/NCS and biopsy in more complicated or unusual cases. (*Id.* at 67-68.) However, she explained that nerve conduction study may not detect all nerve issues, which is why an accurate and thorough medical history is important in these cases. (*Id.* at 68.) In fact, a positive nerve conduction study would, by definition, not be an isolated SFN, because only the large and medium nerves show up on the nerve conduction study. (*Id.* at 68-69, 77, 89-90.) Later in her testimony, Dr. Boylan clarified that SFN is a "clinical diagnosis" that is determined by looking at a patient's history, the time course of the neuropathy, and a clinical examination to look for areflexia, which is "an early sign of" GBS. (*Id.* at 88.) She specifically noted that acute onset suggests an "immune mediated event." (*Id.* at 88.) She explained that some specialty centers will diagnose through a biopsy, however, this is "not routine clinical care," and the lack of a positive biopsy does not necessarily mean the patient does not have SFN. (*Id.* at 89-91.) She also explained that the clinical presentation for SFN would include stabbing, burning, palpitations, dizziness, extreme fatigue, dry mouth, dry eyes, and dysmotility. (*Id.* at 92.)

Dr. Boylan reiterated her view that that "small fiber neuropathy is a form of GBS." (Tr. 77.) She relied on the article, *Sensory Guillain Barré Syndrome and Related Disorders: An Attempt at Systematization*, to explain a proposal to classify this sensory phenomenon into three subtypes under the umbrella of GBS: demyelinating polyneuropathy that would be diagnosed on the basis of electrodiagnostic findings and physical examination; "acute sensory large fiber axonopathy, ganglionopathy," which would also be diagnosed by electrodiagnostic study; and "acute sensory small fiber neuropathy, ganglionopathy," which would likely be characterized by normal electrodiagnostic study. (*Id.* at 79-80 (citing Uncini & Yuki, *supra*, at Ex. 22, p. 1).) Dr. Boylan also noted that the authors document "vaccine-induced neuropathy involving the small fiber nerves." (*Id.* at 81 (citing Uncini & Yuki, *supra,* at Ex. 22, p. 4).) However, Dr. Boylan acknowledged that, unlike petitioner, these patients were ultimately diagnosed though a biopsy. (*Id.*)

Dr. Boylan opined that vaccination, through the mechanism of molecular mimicry, can cause SFN. (Tr. 82.) Although she acknowledged that there is not a consensus in the scientific community as to how vaccines can cause SFN, Dr. Boylan asserts that there is medical literature to support a theory of molecular mimicry between GBS and *Campylobacter jejuni*. (*Id.* at 82-83.) She therefore suggested that it would be "arbitrary" to classify small fibers "in a different category than the traditional . . . acute motor axonal neuropathy and acute sensory axonal neuropathy variants." (*Id.*) She explained that there would need to be some homology between the vaccine and the small fiber cells. (*Id.* at 85.) In addition, she opined that SFN can be immune mediated and would therefore require an "immune system provocation" to trigger such response. (*Id.* at 83-85.)

Dr. Boylan testified that, in her opinion, petitioner "has an autoimmune-mediated small fiber neuropathy, isolated small fiber neuropathy, which is primarily but not exclusively length dependent, caused by the vaccine or vaccines." (Tr. 69.) She noted that, prior to the subject vaccinations, petitioner "had a very striking autoimmune

response to a strep infection" and developed rheumatic fever, "one of the group A streptococcal autoimmune conditions." (*Id.* at 86-87.) Dr. Boylan opined that petitioner had some genetic susceptibility to those conditions, making it more likely that he would develop autoimmune disorders in the future. (*Id.* at 87-88.)

Dr. Boylan relied on petitioner's medical records to explain his condition. (Tr. 92.) She testified that petitioner did not have any symptoms of peripheral neuropathy during his July 28, 2014 appointment. (*Id.* at 92-93 (citing Ex. 2, pp. 9, 12-15).) She noted that, during this appointment, petitioner received both the Hep B and Tdap vaccines. (*Id.* at 94 (citing Ex. 2, p. 15).) She explained that, based on her review of the records in this case, petitioner developed flu-like symptoms roughly 18 hours following his vaccination. (*Id.* at 94-95.) Dr. Boylan explained that these symptoms represent "signs that the inflammatory system has been activated." (*Id.* at 95-96.) However, she stated that petitioner's neurological symptoms, including tingling and numbness, then began two to three days after he received his vaccinations. (*Id.* at 96-99 (citing Ex. 7, p. 4; Ex. 6, p. 2; Ex. 2, pp. 16-17, 19).) Dr. Boylan noted that petitioner's primary care physician then referred to him to a neurologist, Dr. Cholfin, who documented petitioner's symptoms as beginning after he received "the vaccinations." (*Id.* at 99-100.) She acknowledged Dr. Cholfin's note there was no evidence of GBS, and that petitioner's symptoms were "subjective," meaning "the patient feels it, but they can't find anything on exam." (*Id.* at 100-01.) However, Dr. Boylan explained that the neurologist did not test temperature, pinprick, or vibration, which implies that petitioner's paresthesias and numbness was more likely to be considered subjective. (*Id.* at 101-02.) This complication in diagnosing SFN is supported by the medical literature submitted by petitioner. (*Id.* at 102-03 (citing Oaklander & Nolano, *supra*, at Ex. 32).) In addition, Dr. Boylan noted that petitioner's symptoms continued to progress and eventually petitioner began to suffer sensory loss. Accordingly, she suggests the symptoms were not subjective and instead evidence of isolated small fiber neuropathy, specifically an autoimmune mediated case. (*Id.* at 102 (citing Ex. 7, p. 12).)

Dr. Boylan later clarified that petitioner was, in fact, diagnosed with small fiber neuropathy because his treating physician diagnosed neuropathy while noting that there was no medium or large fiber neuropathy, which suggests he believed it was small fiber neuropathy. Additionally, the treating physician proposed a "mechanism which could damage small fibers, immune complex deposition." (Tr. 110-11 (citing Ex. 2, p. 63).) She explained that immune complex deposition is "when combinations of antibodies" and "antigen deposits" get stuck to the vessel walls. (*Id.* at 111.) However, she acknowledged that she is not an immunologist and could not further postulate on "an endogenous antibody cross-reaction." (*Id.*)

Dr. Boylan also addressed petitioner's bloodwork. (Tr. 107-08 (citing Ex. 2, p. 62).) She noted that petitioner's bloodwork showed an increase in ANA, which is associated with a "rheumatologic and autoimmune disease." (*Id.* at 108.) In addition, petitioner's bloodwork showed low complement protein C3, which is "part of the immune inflammatory cascade" and can also suggest an autoimmune disease. (*Id.* at 108-09.)

Dr. Boylan opined that it is fair "to extrapolate medical literature and precedent regarding what we know about the onset of immune-mediated GBS over to immune-mediated small fiber neuropathy." (Tr. 112.) She explained that the time course is similar, and petitioner's illness still followed the "expected trajectory in GBS," despite a latency of two days being "a little short." (*Id.*) Dr. Boylan stated that she did not "see a reason to exclude a small fiber variant" of GBS. (*Id.* at 112-13.) Dr. Boylan explained that onset of symptoms can work on a bell curve, meaning that onset of 48 hours may be fast for some patients while some outliers experience onset of symptoms even faster. (*Id.* at 124.) She explained that an instantaneous response would not suggest an autoimmune mediated phenomenon because that is too fast, however, 48 hours is not outside of the realm of possibility. (*Id.* at 130-31.)

Although she indicated that she is unable to parse which of petitioner's vaccinations is causal, Dr. Boylan opined that it was "probably" the Hep B vaccine, given the fact that hepatitis "is sometimes associated with a small fiber neuropathy." (Tr. 125.) She explained that this theory is based on medical literature that shows a potential molecular connection between hepatitis and polyarteritis nodosa, which is a "multisystemic disease" that is "one of the causes of a small fiber neuropathy." (*Id.* at 128-29.)

f. Dr. Gelfand's in court testimony

Dr. Gelfand testified at the hearing on behalf of respondent. (Tr. 143.) He began his testimony by expressing his opinion that petitioner developed "an acute onset sensory syndrome, and the evidence to support this is overwhelmingly clinical and primarily based on the history without additional supporting diagnostics." (*Id.*) He acknowledged that petitioner did not have any diagnostic tests, "rather than tests that were necessarily done and negative." (*Id.* at 143-44.)

Dr. Gelfand defined small fiber neuropathy as an "impairment of small fiber nerves," which are "either thinly myelinated or unmyelinated small fiber nerves that primarily involve sensation of pain, temperature, and those sensory functions." (Tr. 144.) He explained that there are many causes of SFN, including diabetes, alcohol use, infections, medications, genetics, and autoimmune conditions. (*Id.* at 146-147.) When the cause cannot be determined, the SFN is referred to as idiopathic. (*Id.* at 147.) Specifically, Dr. Gelfand opined that there is no evidence either the Hep B or Tdap vaccine can cause SFN. (*Id.* at 149, 153.) However, Dr. Gelfand noted that "literature establishes associations between several autoimmune conditions" and SFN. (*Id.* at 150-51.) He later admitted that the Hep B virus "is associated and probably causative with certain kinds of peripheral neuropathy, in particular, polyneuropathy, meaning there's a component of large fiber neuropathy, including sometimes this association with vasculitis type processes." (*Id.* at 161.) Specifically, he explained that, while there can be small fiber components "with large fiber polyneuropathies," the relationship between Hep B and SFN is "not as clear or established of an association." (*Id.* at 162.) Dr. Gelfand explained that there have been "antigenic features of the hepatitis B virus that have been implicated as part of the immune response and proposed of the

pathogenesis." (*Id.* at 163.) However, he noted that he does not recall the specific literature concerning the surface antigen that is responsible for this association. (*Id.*) Dr. Gelfand did admit that the Hep B vaccine uses parts of the hepatitis B virus "to create this antigenic response." (*Id.* at 164.)

Dr. Gelfand testified that he considers GBS and SFN to be distinct conditions. (Tr. 147.) However, he acknowledged that there are analogies between the two "that can be important." (*Id.*) He explained that "an isolated small fiber neuropathy is not established as a recognized stand-alone form" of GBS in the current medical literature. (*Id.* at 148.) Dr. Gelfand noted that "the literature postulates whether there can be immune causes of an isolated small fiber neuropathy" and that some of the literature proposes that SFN be considered "an atypical variant" of GBS. (*Id.* at 148, 172.) Dr. Gelfand explained, "I think that can be considered as its own etiologic or pathophysiologic entity, and as sort of separate from what is established about classic [GBS]." (*Id.* at 148.) He also agreed that SFN can be immune mediated. (*Id.* at 172-73.)

Dr. Gelfand noted that petitioner never underwent a skin biopsy or QSART testing.[7] (Tr. 149-50.) He opined that this is significant because, "[t]o diagnose small fiber neuropathy, there needs to be a very strong clinical history, correlated with physical examination, and then additional investigations to support what patterns of nerves are involved." (*Id.*) Although he admitted these tests are not necessary for diagnosis, Dr. Gelfand emphasized that they "increase the confidence in diagnosis." (*Id.* at 150.) He agreed with petitioner's expert that a normal nerve conduction study simply means there is "no evidence of a large fiber neuropathy," and that "[i]n routine clinical practice, small fiber neuropathy is often diagnosed in some form based on clinical history." (*Id.* at 165-66; 168.) He also agreed that "[i]n routine clinical practice, skin biopsies are not commonly ordered." (*Id.* at 167.) He explained that petitioner's "neurologic examinations did not demonstrate evidence of weakness nor was there any clear evidence of impairment of motor function." (*Id.* at 151.) All of petitioner's "[r]eflexes were documented as normal." (*Id.*) In addition, there is "no evidence of an upper motor neuron pattern of involvement to suggest a central nervous system localization." (*Id.* at 152.) Dr. Gelfand did not explicitly opine as to petitioner's diagnosis; however, he explained, "I believe that a small fiber neuropathy is a reasonable phenotypic consideration and is possible, but with the data and available evidence, I cannot give a more specific diagnosis or support a diagnosis to a reasonable degree of medical certainty." (*Id.*) He later agreed that petitioner's treating physician did document sensory symptoms and that "small fiber neuropathy is a reasonable but unconfirmed working diagnosis." (*Id.* 156-57, 187-89.) Dr. Gelfand also admitted that petitioner's treating physician did describe his symptoms as a "vaccine reaction." (*Id.* at 190.)

[7] Dr. Gelfand explained that QSART is a type of testing that analyzes automatic functions of the nervous system and examine responses. (Tr. 150.) He acknowledged that the availability of this form of testing is limited to "a few specialized centers." (*Id.*) As such, Dr. Gelfand suggested that small fiber skin biopsy is the form of testing that would be "most accessible to patients." (*Id.*)

Dr. Gelfand also addressed the time frame proposed by petitioner's expert. (Tr. 153.) He opined that there are two time frames: the first involved petitioner's fever and aching, which began 18 hours after vaccination; and the second is the sensory syndrome that started about 48 hours after vaccination. (*Id.* at 153-54.) He opined that an onset of 48 hours is plausible in some contexts, but the plausibility depends on the time it takes for an immune response to form and cause a reaction. (*Id.* at 159-60.) He also opined that, "[i]n general, it takes several days for an immune-mediated process," and that onset "somewhere between 72 hours to five days would be common in the literature." (*Id.* at 174-75.) Dr. Gelfand agreed that immune mediated SFN would require some sort of immune response. (*Id.* at 181-82.) He further agreed that petitioner's symptoms that developed 18 hours after vaccination were evidence of a "systemic inflammatory response." (*Id.* at 182.)

V. Discussion

a. *Althen* prong one

Under *Althen* prong one, petitioner must provide a "reputable medical theory," demonstrating that the vaccine received can cause the type of injury alleged. *Pafford v. Sec'y of Health & Human Servs.*, 451 F.3d 1352, 1355-56 (Fed. Cir. 2006) (quoting *Pafford v. Sec'y of Health & Human Servs.*, No. 01-0165V, 2004 WL 1717359, at *4 (Fed. Cl. Spec. Mstr. July 16, 2004)). To satisfy this prong, petitioner's theory must be based on a "sound and reliable medical or scientific explanation." *Knudsen*, 35 F.3d at 548. Such a theory must only be "legally probable, not medically or scientifically certain." *Id.* at 548-49. However, petitioners may satisfy the first *Althen* prong without resort to medical literature, epidemiological studies, demonstration of a specific mechanism, or a generally accepted medical theory. *Andreu ex rel. Andreu v. Sec'y of Health & Human Servs.*, 569 F.3d 1367, 1378-79 (Fed. Cir. 2009) (citing *Capizzano v. Sec'y of Health & Human Servs.*, 440 F.3d 1317, 1325-26 (Fed. Cir. 2006)). Scientific evidence offered to establish *Althen* prong one is viewed "not through the lens of the laboratorian, but instead from the vantage point of the Vaccine Act's preponderant evidence standard." *Id.* at 1380.

Petitioner argues that GBS should be considered a "global neuropathy," instead of only a large fiber neuropathy, and, therefore, SFN should be considered a form of GBS. (ECF No. 81 (citing Pan et al., *supra*, at Ex. 17; Seneviratne & Gunasekera, *supra*, at Ex. 20; Yuki et al., *supra*, at Ex. 21; Unciini & Yuki, *supra*, at Ex. 22).) Dr. Boylan submits that, because SFN is a form of GBS, the medical theories that underlie the connections between GBS and vaccines, such as molecular mimicry, can be applied here. (Tr. 82.) Specifically, petitioner notes that respondent's expert has acknowledged that, like GBS, SFN can be immune mediated. (Tr. 7; 160.) Petitioner cites a case in which the special master accepted SFN as a GBS variant that affects small fibers. (ECF No. 81, pp. 23-25 (discussing *Swaiss v. Secretary of Health & Human Services*, No. 15-286V, 2019 WL 6520791 (Fed. Cl. Spec. Mstr. Nov. 4, 2019)). Additionally, the special master concluded that SFN could be immune mediated and, therefore, there was enough evidence to support the conclusion that the Tdap vaccine can cause SFN,

as a variant of GBS. (*Id.*) Dr. Boylan asserts that petitioner's history of autoimmune disorders made him more vulnerable to an immune mediated reaction to the vaccination. (Ex. 10, pp. 6-9; Ex. 29, p. 2.)

Respondent disagrees. Instead, he argues, that petitioner "did not offer a theory of causation that explained how the Tdap and/or Hep B vaccines can cause petitioner's alleged SFN." (ECF No. 83, p. 18.) He further asserts that there is "no evidence to support for Dr. Boylan's opinion that the Hep B and Tdap vaccines could have caused GBS, particularly a small fiber variant of GBS." (*Id.* at 19 (citing Ex. C, p. 9).) In support of this conclusion, respondent points to a 2012 IOM report that "concluded that the 'epidemiological evidence is insufficient or absent to assess an association between hepatitis B vaccine and GBS. . . . The evidence is inadequate to accept or reject a causal relationship between hepatitis B vaccine and GBS.'" (*Id.* at 19-20 (quoting 2012 IOM Report).) Respondent contends that petitioner's theory in this case is "too generalized" to pertain to this specific case and, therefore, petitioner has failed to carry his burden as to *Althen* prong one. (*Id.* at 20 (citing *W.C. v. Sec'y of Health & Human Servs.,* 704 F.3d 1352, 1360 (Fed. Cir. 2013).)

I have previously accepted that the sensory variant of GBS can be caused by flu vaccination. *See Goforth v. Sec'y of Health & Human Servs.*, No. 14-1128V, 2021 WL 6337672 (Fed. Cl. Spec. Mstr. Nov. 19, 2021). However, I have also previously found that the causes of GBS standing alone are not informative of the cause(s) of SFN. *McGill v. Sec'y of Health & Human Servs.,* No. 15-1485V, 2023 WL 3813524, at *26-27 (Fed. Cl. Spec. Mstr. May 11, 2023.) The *McGill* petitioner had a presentation that included SFN arising in the context of preexisting rheumatoid arthritis. *Id*. at *31-33. Especially in that context, the multitude of causes of SFN, including a strong association with rheumatic autoimmune conditions, coupled with the multitude of causes and variants of GBS, confounded petitioner's attempt to rely on analogy to GBS as the primary support for her theory of causation. The literature filed in that case indicated that only a subset of SFN presentations result from tissue-specific dysimmunity that could potentially be compared to GBS. *Id*. at *26. In this case, both parties' experts have provided testimony that accords with that analysis. (Tr. 144-47 (Dr. Gelfand explaining the list of causes of SFN is "very long" and includes a "wide range of injuries"); Tr. 69-70 (Dr. Boylan discussing different forms of GBS with differing nerve involvement); Tr. 87, 147 (both experts discussing causal relationship between autoimmune rheumatic conditions and SFN).) In fact, Dr. Gelfand was also respondent's neurology expert in the *McGill* case.

Here, however, Dr. Boylan has specifically explained, not only that an acute onset suggests an immunologically mediated event, but further that petitioner's own SFN fits the acute, monophasic pattern otherwise seen in GBS. (*Id.* at 88, 112-13.) Dr. Gelfand likewise acknowledges the importance of this distinction in assessing the strength of any analogy to GBS and agrees that petitioner's condition was monophasic and reached a nadir within days to weeks. (*Id.* at 147-48 (indicating that analogy between GBS and SFN can be "important" but stressing the monophasic inflammatory

process targeting antigens in the peripheral (large fiber) nerves involved in GBS); Ex. C, p. 7.)

Though Dr. Gelfand stresses that he would maintain the distinction between SFN and GBS, he agrees on this record that, while SFN does not fall under the umbrella of classic GBS, analogy with respect to the immune causes of GBS may be reasonable and there may be similarities between the causes of GBS and SFN. (Tr. 173.) He suggested that there is at least some degree to which the distinction is semantic. (*Id.* at 147-48, 172.) Regardless of the analogy, Dr. Gelfand agrees that infectious and immune causes are among the proposed causes of SFN. (*Id.* at 146-47.) Both experts also agreed that large fiber polyneuropathies can include small fiber components. (*Id.* at 133; 162.) In that regard, Dr. Boylan opined that it is "arbitrary" to separate cases with only small fiber involvement from "traditional" or large fiber involved neuropathies when determining what pathogenic antibodies may be involved or what may trigger those antibodies. (*Id.* at 82-83.) On this record, that opinion is effectively unrebutted, despite Dr. Gelfand repeatedly noting the distinction between large and small fiber involvement more generally.[8]

Further to that, both experts agree that the hepatitis B virus can cause peripheral neuropathies, most notably a demyelinating large fiber polyneuropathy known as polyarteritis nodosa, but also acute large fiber polyneuropathies more generally. (Ex. 10, p. 3; Tr. 128; 161.) Polyarteritis nodosa is a multisystemic disease, but it is also known to cause SFN. (Tr. 129.) Dr. Gelfand maintains that hepatitis B-related polyneuropathies are a distinct clinical syndrome from GBS with separate supporting associational evidence, but agreed they would share a similar immune mediated mechanism. (*Id.* at 162; 173.) He acknowledged that GBS is a form of acute inflammatory polyneuropathy for which the hepatitis B virus could be a "possible" but "not well established" infectious cause. (*Id.* at 163.) He agreed that antigenic features of the hepatitis B virus are implicated in the immune response pathogenic for polyneuropathies. (*Id.*) Thus, Dr. Boylan opines that molecular mimicry, which has otherwise been implicated with respect to GBS, is also a likely explanation for the causal connection between the hepatitis B virus and peripheral neuropathies. Further, she opines that this also supports molecular mimicry between the hepatitis B surface antigen contained in the Hep B vaccine and the peripheral nerves as a cause of SFN. (*Id.* at 133; 162-64.) Dr. Boylan explained that extrapolating from hepatitis B-induced peripheral neuropathies is reasonable because molecular mimicry works like a lock and key. (*Id.* at 133.) Where the same proteins and structures are at issue, the fit between the lock and key will be the same. (*See id.*) Although Dr. Gelfand does not agree that

[8] One factor discussed in *McGill* is that the small fibers are largely unmyelinated whereas myelin is often identified as the key target of GBS injury when seeking to implicate vaccination as a cause. 2023 WL 3813524, at *30. The most commonly discussed form of GBS is acute inflammatory demyelinating polyneuropathy. (Tr. 70.) However, as both experts indicated in this case, some of the small fibers are lightly myelinated. (*Id.* at 61, 144.) Accordingly, the distinction is not necessarily dispositive on its own. Moreover, when specifically asked about cross-reaction between campylobacter antigen (a known cause of GBS) and myelin tissue, Dr. Gelfand was careful not to limit his response to myelin specifically, noting that "different antigens have been identified and postulated within the spectrum of Guillain-Barre syndrome, you know, depending on whether it's myelin or axonal or others." (Tr. 181.)

the Hep B vaccine is a *recognized* cause of SFN, he acknowledged that the Hep B vaccine uses "parts of the virus" to create an antigenic response and demurred on any further specifics. (*Id.* at 149, 164.) Therefore, Dr. Boylan has provided largely unrebutted expert testimony opining that hepatitis B surface antigen, as contained within the Hep B vaccine, can cause peripheral neuropathies, including SFN.

In light of all of the above, I find Dr. Boylan's theory of causation to be sound and reliable within the context of this record. Therefore, petitioner has preponderantly established that the Hep B vaccine can cause SFN. This is not necessarily surprising given that prior cases have concluded that the Hep B vaccine can cause both GBS and SFN. *See, e.g.*, *Shaw v. Sec'y of Health & Human Servs.*, No. 01-0707V, 2013 WL 2897425, at *15-16 (Fed. Cl. Spec. Mstr. May 24, 2013) (accepting that the Hep B vaccine can cause SFN via molecular mimicry despite a lack of direct evidence); *Peugh v. Sec'y of Health & Human Servs.*, No. 99-638V, 2007 WL 1531666 (Fed Cl. Spec. Mstr. May 8, 2007) (finding as part of the "Hepatitis B-Neurological Demyelinating Omnibus Proceeding" that the Hep B vaccine can cause GBS).

b. *Althen* prong two

The second *Althen* prong requires proof of a logical sequence of cause and effect, usually supported by facts derived from a petitioner's medical records. *Althen*, 418 F.3d at 1278; *Andreu*, 569 F.3d at 1375-77; *Capizzano*, 440 F.3d at 1326; *Grant v. Sec'y of Health & Human Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992). In establishing that a vaccine "did cause" injury, the opinions and views of the injured party's treating physicians are entitled to some weight. *Andreu*, 569 F.3d at 1367; *Capizzano*, 440 F.3d at 1326 (explaining that "medical records and medical opinion testimony are favored in vaccine cases, as treating physicians are likely to be in the best position to determine whether a 'logical sequence of cause and effect show[s] that the vaccination was the reason for the injury'" (alteration in original) (quoting *Althen*, 418 F.3d at 1280)). Medical records are generally viewed as particularly trustworthy evidence because they are created contemporaneously with the treatment of the patient. *Cucuras v. Sec'y of Health & Human Servs.*, 993 F.2d 1525, 1528 (Fed. Cir. 1993).

However, medical records and/or statements of a treating physician's views do not *per se* bind the special master to adopt the conclusions of such an individual, even if they must be considered and carefully evaluated. *See* § 300aa-13(b)(1) (providing that "[a]ny such diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court"); *Snyder ex rel. Snyder v. Sec'y of Health & Human Servs.*, 88 Fed. Cl. 706, 745 n.67 (2009) (explaining "there is nothing . . . that mandates that the testimony of a treating physician is sacrosanct—that it must be accepted in its entirety and cannot be rebutted"). As with expert testimony offered to establish a theory of causation, the opinions or diagnoses of treating physicians are only as trustworthy as the reasonableness of their suppositions or bases. The views of treating physicians should also be weighed against other, contrary evidence also present in the record, including conflicting opinions among such individuals. *Hibbard v. Sec'y of Health & Human Servs.*, 100 Fed. Cl. 742, 749 (2011) (concluding that it is not

arbitrary or capricious for a special master to weigh competing treating physicians' conclusions against each other), *aff'd*, 698 F.3d 1355 (Fed. Cir. 2012); *Caves v. Sec'y of Health & Human Servs.*, 100 Fed. Cl. 119, 136-37 (2011), *aff'd*, 463 Fed. App'x 932 (Fed. Cir. 2012); *Veryzer v. Sec'y of Health & Human Servs.*, No. 06-522V, 2011 WL 1935813, at *17 (Fed. Cl. Spec. Mstr. Apr. 29, 2011), *motion for review denied*, 100 Fed. Cl. 344 (2011), *aff'd per curiam sub nom. Veryzer v. United States*, 475 Fed. App'x 765 (Fed. Cir. 2012).

Respondent raises two challenges to petitioner's claim under *Althen* prong two. (ECF No. 83, pp. 20-23.) First, respondent contends that petitioner did not actually suffer SFN. (*Id*. at 21-22.) And, second, the treating physician opinions in the case were limited to noting a temporal association and "none of petitioner's treating physicians could explain how the vaccinations can cause the symptoms alleged by petitioner."[9] (*Id.*) Ultimately, neither argument is persuasive.

During the hearing, both experts agreed that petitioner's treating physician diagnosed him with SFN. (Tr. 110-11; 189-90 (citing Ex. 2, p. 63).) In addition, both experts ultimately agreed that a biopsy is not necessary to diagnosis someone with SFN.[10] (*Id.* at 67-68; 150.) Both experts also agree that SFN will not show up on nerve conduction studies because these only show large nerve dysfunction. (*Id.* at 89-90; 165-66, 168-69.) Ultimately, Dr. Gelfand agreed on respondent's behalf that petitioner experienced a sensory syndrome with neuropathic features for which SFN is a "possible" diagnosis or a reasonable, but unconfirmed, working diagnosis. (Tr. 152, 156-57.) Considering the record as a whole, including the treating physician's diagnosis, Dr. Boylan's opinion affirmatively endorsing the SFN diagnosis, and Dr. Gefland's more tentative partial agreement, I conclude that there is preponderant evidence that petitioner suffered SFN.

Further to this, petitioner experienced an acute, post-vaccination reaction that included fever, headache, and muscle and joint aches. (Ex. 7, p. 3.) On respondent's behalf, Dr. Gelfand agreed that this is evidence of a systemic inflammatory reaction. (Tr. 182.) In that regard, petitioner's treating neurologist, Dr. Cholfin, accepted that petitioner suffered a vaccine reaction leading to paresthesia. (Ex. 2, p. 25.) Dr. Boylan also noted that petitioner's bloodwork showed an increase in ANA, which in isolation would be inconsequential, however, when it is associated with a history of autoimmune conditions, is evidence of an ongoing autoimmune response. (Tr. 108 (citing Ex. 2, p. 62).) In addition, petitioner's bloodwork showed low complement protein C3 which is "part of the immune inflammatory cascade," and can also suggest an autoimmune disease. (*Id.* at 108-09.) In fact, respondent himself distinguishes petitioner's initial presentation from the typical, months-to-years development of SFN. (ECF No. 83, p. 21.)

[9] Part of respondent's argument, to be sure, is the timing of onset. (ECF No. 83, pp. 21-22.) However, this is addressed separately under *Althen* prong three.

[10] A biopsy is the gold standard for diagnosing SFN, however that does not mean it is absolutely required to diagnose SFN in all cases. (Gibbons, *supra*, at Ex. 16, p. 5.) In addition, "history and examination" are important diagnostic tools. (*Id.* at 2.)

Petitioner's treating physicians repeatedly characterized his symptoms as a vaccine injury throughout his treatment and noted a suspected "immune complex deposition as reaction to vaccine." (Ex. 2, pp. 49, 53, 61-62.) While respondent challenged the value of this explanation in his prehearing brief (ECF No. 83, p. 22), petitioner's expert, Dr. Boylan, confirmed during the hearing that the immune complex deposition cited by the treating physicians is consistent with both the accepted mechanisms of causation for SFN (Tr. 110-12) and the mechanism by which the hepatitis B antigen is understood to cause peripheral neuropathy in the form of polyarteritis nodosa (Tr. 128-30), though immune complex deposition is not her own theory of causation. Dr. Gelfand likewise identified "vasculitis type processes" as "sometimes" present among the different immunologic causes of polyneuropathy. (Tr. 161-62.) Petitioner's medical records also confirm that his physicians considered, but rejected, the possibility that petitioner's neuropathic symptoms were related to the type of rheumatic conditions that can otherwise be associated with SFN. (Ex. 2, pp. 19, 49.) In fact, this is a point stressed by respondent. (ECF No. 83, p. 22 (asserting that petitioner "did not have other known common causes of small fiber neuropathy").) Thus, I do not agree that the treating physicians' causal opinions were limited to noting temporality.

For the reasons discussed above, petitioner has preponderantly established that there is a logical sequence of cause and effect between his vaccination and his SFN.

c. *Althen* prong three

The third *Althen* prong requires establishing a "proximate temporal relationship" between the vaccination and the injury alleged. *Althen*, 418 F.3d at 1278. That term has been equated to the phrase "medically-acceptable temporal relationship." *Id.* at 1281. A petitioner must offer "preponderant proof that the onset of symptoms occurred within a time frame for which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation-in-fact." *de Bazan v. Sec'y of Health & Human Servs.*, 539 F.3d 1347, 1352 (Fed. Cir. 2008). The explanation for what is a medically acceptable time frame must also coincide with the theory of how the relevant vaccine can cause an injury (*Althen* prong one's requirement). *Id.* at 1352; *Shapiro v. Sec'y of Health & Human Servs.*, 101 Fed. Cl. 532, 542 (2011); *Koehn v. Sec'y of Health & Human Servs.*, No. 11-355V, 2013 WL 3214877, at *26 (Fed. Cl. Spec. Mstr. May 30, 2013), *aff'd*, 773 F.3d 1239 (Fed. Cir. 2014).

Petitioner's testimony and medical records support the conclusion that petitioner's sensory symptoms developed 48 hours after he received his vaccinations. Petitioner received his Hep B and Tdap vaccines on Monday, July 28, 2014. (Ex. 1.) On July 31, 2014, petitioner called his doctor and complained that he had developed flu-like symptoms and that he was feeling generally lethargic and had no energy. (Ex. 6, p. 2.) Petitioner explained that he had felt numbness in his hands since Tuesday and that believed he was experiencing an autoimmune reaction to the Hep B vaccine. (*Id.*) Later that same day, petitioner returned a call to Dr. Kim-Hwang and indicated that his

fever had resolved, but he still felt "generalized lethargy / low energy." (*Id.*) On August 1, 2014, petitioner presented to Dr. Kim-Hwang for an urgent care visit. (Ex. 2, pp. 16-17.) Petitioner explained that he had developed a fever one day after receiving his vaccinations, and then he had developed numbness and tingling on his left side. (*Id.*) Finally, on August 4, 2014, petitioner presented to neurologist Dr. Cholfin. (Ex. 2, p. 20.) Dr. Cholfin also noted that petitioner developed flu-like symptoms one day after receiving the vaccines. (*Id.*) He described that petitioner's fever broke the following day and, soon after, petitioner developed numbness and tingling in his hands and feet. (*Id.*) Petitioner clarified this timeline at the hearing by explaining that his flu-like symptoms developed one day after he received the vaccines, and his sensory symptoms began when his fever broke very early (between 4:00 AM and 5:00 AM) Wednesday morning. (Tr. 21.)

Dr. Boylan opined that the onset timeline for GBS could be used as the onset timeline for SFN. (Ex. 10, p. 3.) She noted that there was usually a delay in post vaccine related neurological syndromes "because the immune response system is a complex cascade involving many different cells and chemical messengers." (*Id.*) During her testimony, Dr. Boylan explained that 48 hours was quick for a vaccine induced neurological reaction, but "biology tends to work in bell curves," meaning that there may be outliers who experience reactions faster than one would normally associate with a vaccine reaction. (Tr. 124.) She acknowledged that there is a cut off on the left side of the bell curve and that instantaneous reactions would not support an autoimmune mediated response; however, she opined that 48 hours was not an unreasonable timeline. (*Id.* at 130-31.) For example, among the series of post-vaccination SFN cases reported by Souayah et al., two had onset occurring within 24 hours of vaccination. (Souayah et al., *supra*, at Ex. 25, p. 3.) Petitioner's own primary care physician, Dr. Kim-Hwang, initially considered whether petitioner might have had GBS and, in weighing that possibility, felt that the timing of onset was "atypical" but "not impossible." (Ex. 2, p. 19.)

Dr. Gelfand did not necessarily disagree with Dr. Boylan. He noted that there are two different time frames in this case: the first involves petitioner developing flu-like symptoms within 18 hours and the second involves petitioner developing sensory symptoms within 48 hours. (ECF No. 83, p. 24 (citing Ex. H, p. 3); Tr. 153-54.) He opined that, while "48 hours or less is very quick for traditional adaptive immune responses," he acknowledged that where exactly the line is drawn is somewhat arbitrary. (Tr. 159-60.) He opined that, with what little is known about small fiber neuropathies, he could not explicitly state the appropriate temporal association, though he did explain that between 72 hours and 42 days would not be unreasonable. (*Id.* at 160.) He opined that all that matters is that there is enough "time for an immune response to form." (*Id.*) However, even when asked directly, at no point did Dr. Gelfand opine that 48 hour falls below that threshold. Instead, he stated that a cut-off "somewhere around three days is reasonable" and disclaimed any opinion that a 48-hour onset is impossible. (Tr. 174-75.)

The Federal Circuit has cautioned against using "hard and fast" deadlines between vaccination and onset of symptoms. *Paluck v. Sec'y of Health & Human Servs.*, 786 F.3d 1373, 1383 (Fed. Cir. 2015). In light of all of the above, petitioner has shown by preponderant evidence that there was a proximate temporal relationship between his vaccination and development of SFN.

d. Factor unrelated to vaccination

Once petitioner has satisfied his own burden pursuant under the *Althen* test, the burden shifts to respondent to demonstrate that petitioner's injury was caused by factors unrelated to vaccination. § 300aa-13(a)(1)(B); *Deribeaux ex rel. Deribeaux v. Sec'y of Health & Human Servs.*, 717 F.3d 1363, 1367 (Fed. Cir. 2013). In this case, Dr. Gelfand confirmed during the hearing that he is not proposing any alternative explanation for petitioner's condition. (Tr. 171-72.)

VI. Conclusion

Accordingly, for all the reasons described above, I find that petitioner is entitled to compensation. Specifically, I find that petitioner has established by preponderant evidence that he developed small fiber neuropathy as a result of his hepatitis B vaccination. A separate damages order will be issued.

IT IS SO ORDERED.

s/Daniel T. Horner
Daniel T. Horner
Special Master